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S.R.T.M. University Nanded & Dr. B.A.M. University, Aurangabad

# APPLIED PARASITOLOGY -I

## (Parasitic Protozoa and Platyhelminthes)

For UG and PG Students


**Authors**

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# PROLAUGE

Text book of Applied Parasitology -I is written as per the As per Choice Based Credit System pattern of S.R.T.M. University Nanded & B.A.M. University, Aurangabad for the Undergraduate and Post graduate students. In this book which is mainly devoted to students of Basic Science, Medical Institute concentrate on those animals which can infect Human being (*Homo sapience*) causing diseases with diversified virulence. Some of these parasites may be fatal such as Plasmodium species causing malaria disease or the larvae of the cestode, *Echinococcus granulosus* which causes Hyatidosis or Hydatid cysts which may cause prophylactic shock and consequently a sudden death. Other parasites are with less effect or may even commensally such as *Entamoeba coli*, infecting human intestine. Anyhow, generally parasites may cause little damage but some time event the commensals turns in to pathogenic. Moreover, studying parasites are not only on its pathological problems but includes all aspect of parasite species, its classification, morphology, ultra-structure, life cycle physiology, epidemiology, modes of transmission, modes of diagnosis their control or, proposed treatment, and the prevention strategy.

# APPLIED PARASITOLOGY-I

## PARASITIC PROTOZOA AND PLATYHELMINTHES

## CONTENTS

# 01 PARASITIC PROTOZOA AND PLATYHELMINTHES

## 1.1. INTRODUCTION OF PARASITOLOGY

Definition of Parasitology

A branch of biology dealing with parasites and parasitism especially among animals.

1) Medical parasitology traditionally has included the study of three major groups of animals, parasitic protozoa, parasitic helminths (worms), and those arthropods that directly cause disease or act as vectors of various pathogen.
2) A parasite is a pathogen that simultaneously injures and derives substances from its host.
3) Some organism called parasites are actually commensals, in that they neither benefit nor harm their host.
   (ex- *Entamoeba Coli*).
4) The diseases caused by parasite constitute major human health problems throughout the world. (e.g. *Ascaris Lumbricoides*).
5) A misconception about parasitic infections is that they occur only in tropical area.
6) Although most parasitic infections are more prevalent in the tropics, many people in temperate and subtropical area also become infected & visitors to tropical countries. May return with a parasite infection.
7) The unicellular parasites (protozoa) & multicellular parasites (*Helminthus arthropods*) are antigenically & biochemically complex are there life histories the pathogenesis of diseases they cause.

### 1.1.1. Parasitism

In this type of association one organism is totally dependent on other host for food and shelter and always cause some harm to host which may be slight or more. E.g. Worms in digestive tract.

## 1.1.2. Types of Parasites

The following are the various types of parasites. An organism which depends upon another organism for nutrition & protection is called parasite.

### 1.1.2.1 Ectoparasite:

These parasites which live on the surface of their host.

***Examples:*** *Hydramoeba, hydraxeno*, feeding on the ectodermal cells of Hydra and *Ichthyopthrius multifilis*, burying in the epidermis of fresh water fishes, are the ectoparasitic protozoa.

### 1.1.2.2. Endoparasite:

Those parasites which live in the internal tissues of the host inside the body of their hosts, these are divided into four categories

#### i) Parasites of the Mouth:

The endo parasite which reside in the mouth cavity of the hosts in man Entamoeba gingivitis and Trichomonas Tenax are found in pockets between the Gums and teeth.

#### ii) Parasites of digestive Tract:

Those endoparasite which dwell inside the lumen of the alimentary canal of the hosts.

Ex. *Giardia laumbia* a parasitic flagellate, *Entamoeba histolytica*, a parasitic *amoeba, Isopora, hominis*, a parasitic *coccidian, Balantidium coil*, a parasitic ciliate are all intestinal endoparasites of man.

#### iii) Parasites of the Genital Tract:

Those endoparasites which inhibit the genital tract of the hosts. In human female, Trichomonas Vaginalis lives in vagina.

#### iv) Parasites of body tissue:

Those parasites which live within the tissue of the hosts and may enter through the skin or from the digestive tract. Species of *Trypanosoma, Leishmania, plasmodium* and Vertebrates, Spp, of *Eimeria* and *Isospora* occur in the epithelial living of the gut of their respective host.

#### v) Hyper Parasite:

These are examples of hyper parasites protozoa i.e. protozoa parasitizing other species of parasitic protozoa for instance the oplimid (zelleriella) which lives in the frog's intestine is parasitisized by a certain *amoeba. Nosema notabilis parasitilizes sphanespo*ra polymorphs which is a parasite of urinary bladder of toad fish.

vi) Pathogenic Parasites:

Most of the parasitic protozoan do not cause disease conditions in their host except producing minor symptoms on the other hand, certain parasite acts as diseases causing organism man and other animals such parasites are reffered to as pathogenic parasites important pathogenic parasites of man are *Leishmania donavani, T. gambiense, plasmodium vivax, E. histolytica* etc.

### 1.1.3. Types of Host

Host: An organism which supplies food and shelter to the parasite is known as the host the host harbours the parasite.

Host-Parasite: The parasite and the host establish close association between them. The parasite lives partly or wholly at the expense of its host. During its life the parasite by absorbing the nutrients from the host body devitalizes it and sometimes release certain toxic substances which are injurious to host.

Definitive Host: It harbours the adult state of the parasite or where the parasite adopts sexual reproduction for many of the human parasite man acts as the definitive host.

Intermediate Host: It harbours the larval stages of the parasite or where the parasite undergoes asexual reproduction, man is the intermediate host for the malarial parasite. In some cases, the Larval development of the parasite is completed in two different intermediate host which are then referred to as first or primary intermediate host and second or secondary intermediate host.

Carrier hosts or paratenic hosts: In some hosts the parasite does not undergo any developmental changes but instead remain viable. These help in transforming the parasite from one host to another and therefore are called carrier hosts or paratenic hosts.

Reservoir host: Reservoir hosts store the parasitic forms in their body. The parasite does not infect this host but stay dormant for some time & infects fresh hosts.

### 1.1.4. Vector

The carrier of the disease-causing germs from one host to another host are called vectors usually the insects are vector of diseases.

### 1.1.5. Host Parasite Relationship

i) The parasitism affects both the parasite and the host.

ii) A sort of mutual adaptation and compromise is developed in order to maintain relationship of the parasite & the host.

iii) It is harmful to internal parasite to cause the death of the host and it is disadvantages to external parasites to cause irritation resulting in the death of the host.

iv) Host parasite relationship is a type of symbiotic relationship between organisms of different species, the parasite benefits at the expense of the host, we have different type include.

1) Commensalism: The association of two different species or organism in which one is benefited and the other is neither benefited nor harmed (e.g. nonpathogenic intestinal protozoa).

2) Mutualism: The relation is benefit to both associates.

3) parasitism: The relation in which parasite is benefit & host is harm.

a) Effect on the Host:

i) A parasite may be non-pathogenic it may have no noticeable ill effects on the hosts.

ii) The effect on the host may be due to a variety of factor.

iii) The parasite robs the host of food materials.

iv) In serious cases, there may be damage to walls of the organs to which the parasites.

v) The eggs and larval penetration and their encystment in various organs result in serious effects on the host.

vi) In a well-polished host relationship, the host protect itself against the injurious effects of the parasites, by producing antibodies to neutralize by produce of the parasite and by making its blood forming & by making tissue repairing mechanism on a strong efficient footing.

b) Effect on the Parasite:

The parasitism broadly speaking is very specialized type of life and the parasite in persuasion of successful parasitism have undergone prefound changes and modification in order to adopt fully inside the host.

c) Parasite of Man:

i) It has been estimated that there are about 500 parasites of common man alone.

ii) Among the notable external parasites are the blood sucking mosquitoes, houseflies, leeches, ticks, mites, bugs and lice etc.

iii) Besides many protozoans endoparasites, several kinds of worms attack man.

iv) Invasion of the body by worms is the most common disease in the world today and nearly a million people suffer from it.

v) In some tropical countries over 90% of the people are infected.

vi) In the USA despite its remarkable progress in sanitation 18 million people are infected with pinworms, 3 million with ascaris 80 million with trichine worms and many others with hook worms and tape worms.

### 1.1.6. Host Specificity:

**Definition:** The resistance and immunity are also associated with the limitation of parasites to particular hosts which is known as hosts specificity.

- They can develop in a variety of hosts survival of parasite in a host two factors -
  - Easy and dependable means of dispersal of progeny.
  - Ability of parasite to live in the host after ingestion.
- These two factors determine the presence of a particular parasite in particular host.
- In strange host where these two conditions are not fulfilled the parasite wanders aimlessly.
- The hook worms of man enter the blood vessels and through blood vessels they reach the lungs and intestine.
- The larva of pig tape worm enters in muscles of normal pig-host and in man they blunder into brain eye.

## 1.2 Modern progress against parasitism:

**i) Best Protection:**

- The through study of the life cycle histories & parasites provides the best means for protection against them.
- The great progress has been made in Africa against the frightful sleeping sickness protozoans' parasite by eliminating its vector the tse-tse fly.
- The similarly the source of malaria has been wiped out in several countries.

- The naturally the greatest stride have been made in the most liberate countries where mass education and higher standards of living have enabled man to wage a scientific welfare against these age-old rivals.

ii) Avoiding Contamination:

- In most modern countries contaminated by parasites has been reduced to a bare minimum by adopting modern methods of water purification and the sewage disposal routine meat inspection minimizes the danger of infection by parasitic worms such as beef and pig tape worms.
- The new insecticides have effectively reduced the hazards of insect parasite.
- Wide spread public health education has also contributed to this effect.

iii) New Drugs:

- The several new wonder drugs have been developed of continuance the menace of parasites.
- One of this miracle drug in dithiazamine iodide used successfully against the variety of intestinal worms.
- This drug neutralizes specific worm enzymes serving to release oxygen from the food of parasite.
- Thus, oxygen supply is cut off and the worm soon dies from suffocation.
- Some of the other revolutionary modern drugs are bephenium, piperazine and pyrvinium etc.

## 1.3. Scope and branches of Parasitology:

It is one of the widest branches of the whole complex of biological science and medical, veterinary and psycopathological microbiology, mycotology, virology, protozoology, heminthology, entomology and archenology are all separate divisions of parasitology science in its widest sense.

All these divisions deal with parasitism and consequently with pathogenesis and immunity, they consider not only the evolution of host and parasite but also the evolution of parasite host system as a whole, with interrelationship of the components of the system.

# 02 PARASITIC PROTOZOA

Contents



## 2.1.1. Introduction:

Protozoal parasite consists of a single "cell like unit" which is morphologically and functionally complete the difference between protozoa and metazoan are as follows.

### Protozoa:

1) Unicellular.
2) A single cell like unit.

3) A single cell performs all the functions reproduction, digestion, respiration, excretion, etc.

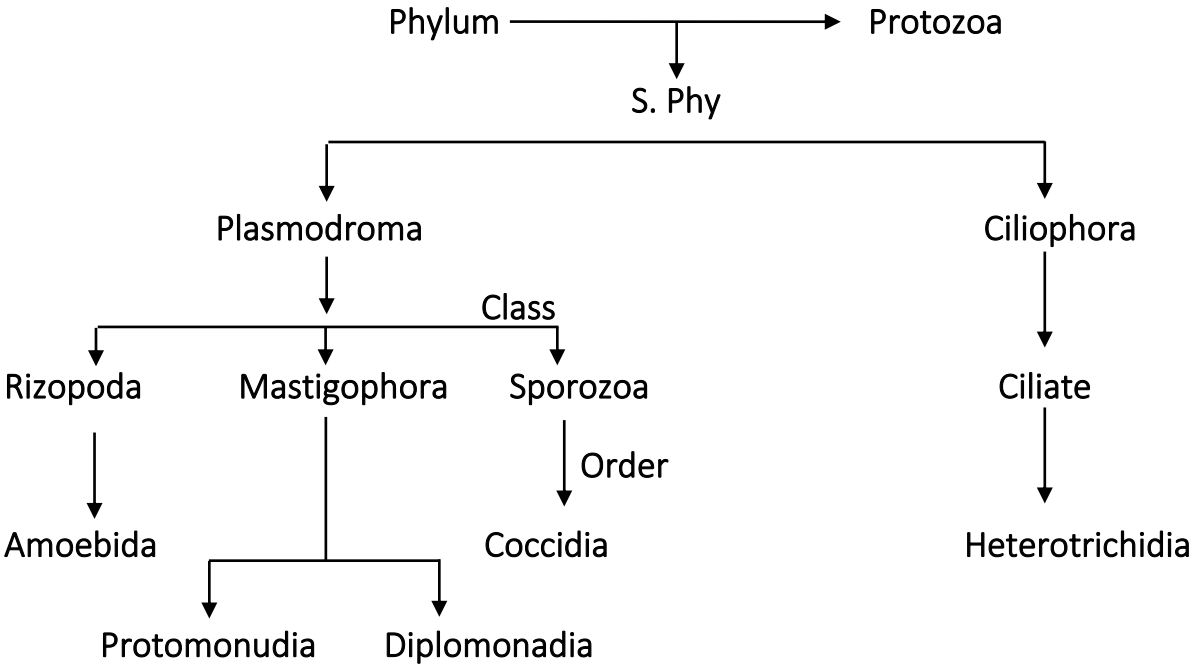
**Metazoa:**

1) Multicellular.
2) A number of cells making up a complex individual.
3) Each special cell performs a particular function.

In parasitic protozoa size, shape, variable body with single nucleolus and cytoplasm generally, single nucleus is present but in ciliates two; one micro and macro nucleus, cytoplasm divided into two; ecto and endoplasm.

### 2.2.1. Classification of protozoa.

According to the scheme conventionally in use of the protozoal parasites of man are classified as follows:




### 2.2.2. General Organization of Parasitic Protozoa.

**Morphology:** The structure of protozoal cell is composed of –

(i) Cytoplasmic body (ii) Nucleus

**Cytoplasm :**

**a) Ectoplasm:** The external hyaline portion it's function is protective locomotion and sensory.

**b) Entoplasm:** The internal granular portion its function is nutritive and reproductive.

**Structures Developed from Ectoplasm:**

i) Structures of organells of Locomotion:

**a) Pseudopodia:** Prolongation of temporary ectoplasmic process, seen in Rhizopodia.

**b) Flagella:** Long, delicate, thread like filaments seen in zoomastigophora.

**c) Cilia:** Fine needle like filaments covering the entire surface of the body been in ciliata.

**ii) Contractile Vacuoles:** Situated inside the endoplasm, excretory function.

**iii) Rudimentary digestive organ:** Such as cytostome (cell mouth) & cytopharynx seep in balantidium coli.

v) **Cyst Wall:** A thickened resistant wall, seen in the cystic stage.

**Nucleus:**

It is the most important structure as it controls the various function and regulates reproduction. It's structure comprises the following main parts: -

I. It is bounded externally by a well-defined nuclear membrane.
II. Made up of network of lining enclosing within it the nuclear sap.
III. Chromatin granules, lining the inner side of the nuclear membrane or appearing as condensation on lining threads.
IV. Karyosome situated inside the nucleus either or peripherally.

**Encystment**

The protozoal parasite possesses the property of being transformed from an active (trophozoite) to an inactive stage, loosing it's power of mortality and enclosing itself within a tough wall the protoplasmic body thus formed is known as a cyst. At this stage, the parasite loses its power of growth and the multiplication.

**Reproduction**

The protozoal parasite may exist in two stages, trophozoite and cyst as in intestinal flagellates and amoebae. In such cases the parasite multiplies only

in the trophic stage, the methods of reproduction of or multiplicate among the parasite protozoa are of the following types:

1) Asexual multiplication / reproduction

i) By simple binary fission: In this process the individual parasite divides either longitudinally or transversely into two more or less equal parts. Before division all the structures are duplicated.

ii) By multiple fission or schizogany: In this process more than two individual are produced as in plasmodia. The nucleus of parent cell as first undergoes repeated division which are then surrounded by the cytoplasm when the multiplication is completed, the parasitic body or the schizont ruptures and liberates these daughter individuals which in their turn repeat their life cycle.

2) Sexual reproduction

i) By Conjugation: In this process a temporary union of two individuals occurs during which time interchange of nuclear material take places. Later or the two individuals separates, each being rejuvenated by the process as in ciliates.

ii) By Syngamy :- In this process, sexually differentiated cells, called gametes, unite permanently and complete fusion of the nuclear material take place, the resulting product is then known as a zygote, as in plasmodia.

### 2.2.1 *Trypanosoma gambiense.*

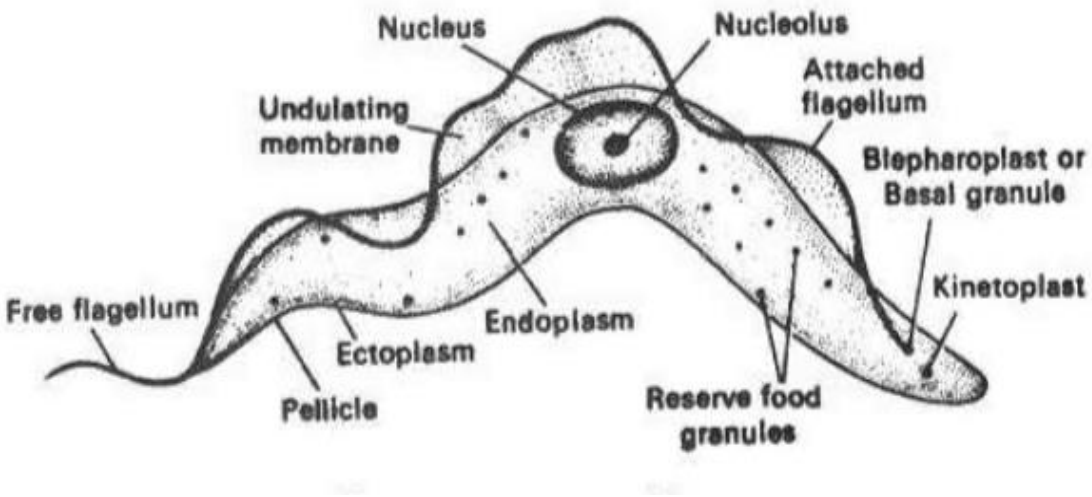
Systematic Position

| | | |
|---|---|---|
| Phylum | - | Protozoa |
| Sub-Phy. | - | Plasmodroma |
| Class | - | Mastigophora |
| Order | - | Protomonadina |
| Family | - | Trypanosomatidae |
| Genus | - | Trypansoma |
| Species | - | *T. gambiense* |

Geographical Distribution

Trypanosoma is found in the West and Central Africa at many places, particularly in Nigeria and congo.

Morphology




The adult Trypanosoma has an elongated dorso ventrally flattend. It is cellular sub-microscopic organism. The body is 10 to 15 microne in length and 1 to 4 microne in width. The adult trypanosome has an elongated body with pointed end it has spherical nucleus containing distinct nucleous flagellum which arises in the body posterior from a basal granuls. The flagellum runs along with the free border in the cytoplasm below the

nucleus there are two small granules, known as basal granules and parabasal body which are usually connected together by rhizoplast (a thin filament).

### Flagellum

Trypanosoma gambiense is an unflagellate because is bears single, strong locomotary flagellum. It extends through the length of body.

### Undulating membrane

The flagellum is remains attached to pellicle by means of a thin membrane called undulating membrane. It is adaptation for the locomotion in the body.

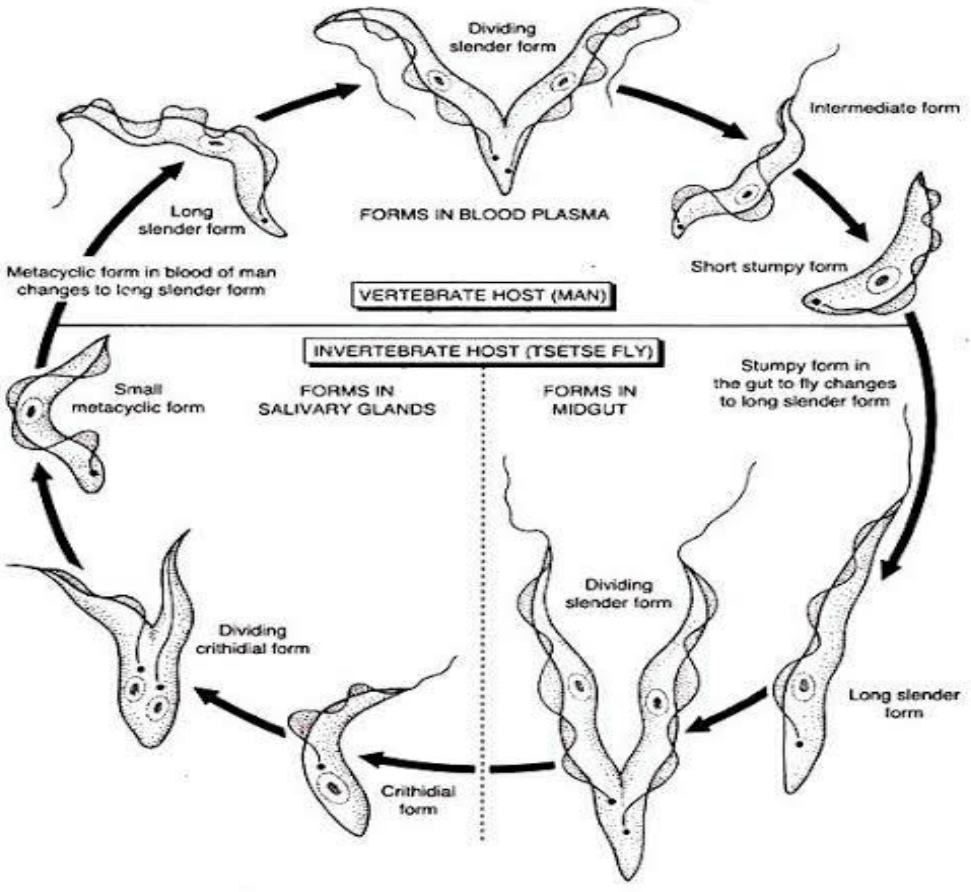
### Pellicle

The body of Trypanosoma is surrounded by thin elastic firm and protecting cover called pellicle due to which the body shape of typanosoma is constant.

### Cytoplasmic material

The body cytoplasm is divided into ectoplasm and endoplasm, cytoplasm consist of several scattered greenish particles called volutin granules.

### Life-Cycle



Trypanosoma is a diagenetic parasite and completes it's life cycle in two host. Man is primary or definite host and secondary or intermediate host is an invertebrate, a Tse-Tse fly which is on bitting inset, Trypanosoma gambiense is transmitted to man through the bite of Tse-Tse fly. Glossina palpalis which serves as the main carrier of vector of species.

## Mode of Transmission Life-Cycle in Man:

It occurs in two methods, transmission in man and transmission in Tse-Tse fly. The infective form is metacyclic form. This is present in the salivary gland of Tse-Tse fly. These flies liberate numerous trypanosomes into blood stream of man. Firstly, it is puncturing the skin surface by with the help of probasis this metacyclic forms with the help of it this feed in the blood and converted into slender forms this long slender form undergoes Logitudinal binary fusion the various forms are produce, but stumpy form this are called intermediate form. This form is infective to Tse-Tse fly some of the stumpy form are converted into long slender Trypannosomes this undergo repeated longitudinal binary fission and causes various symptoms are Trypanosomiasis.

In man trypanosome undergo it's life-cycle extra cellularly ile its development take place in blood plasma and not in blood cells.

## Mode of transmission in Tse-Tse fly:

Where Tse-Tse fly sucks the blood of infected person, short stimpy forms are also taken into the midgut of an insect. The long slender forms multiply by longitudinal binary fission. The large number of long slender forms are produced after several days. They pass in the salivary glands through oesophagus, mouth parts, some of the long slender forms. Multiply in the human of the salivary glands and transforms into the slender trypanosome forms known as metacyclic forms.

In Trypanosoma gambiense only two development forms are found this are crethedial forms generally crethedial forms found in the invertebrate host only i.e. Tse-Tse fly. The remaining forms are rarely forms in this species.

## Pathogenicity

T. gamblense cause a disease is known as Trypanosomiasis or African sleeping sickness and Gambian fever. When the parasite live in blood they poison the blood with their waste products, causing fever recurring at regular intervals, anaemia, weakness, enlarged gland.

Finally they reaches into cerebrospinal fluid that surrounds the brain and spinal chord of the patient who loses convausness and sleeping sickness goes to foetal end. The bite of Tse-Tse fly causes irritation and itching which gives painful inflammation.

## Diagnosis

The diagnosis of African Tripanosomiasis is made through laboratory methods because the clinical features of infections are not sufficiently specific. The diagnosis rests on finding the parasite in body fluid or tissue by microscopy. The parasite load in T. rhodesiense infection is substantially higher than the level in *T. gambiense* infection.

The classic method for diagnosing *T. gambiense* infection is by microscopic examination of lymphy node aspiratex usually from a posterior cervical node. It is often difficult to detect *T. gambiense* in blood.

Trypanosomoes can often be observed in cerebrospinal fluid in persons with second stage infections.

## Prophylaxis

There is no vaccine or drug for prophylaxis against African trypanosomiasis. Preventive measures are aimed at minimizing contact with tst-tse flies. Local residents are usually aware of the areas that are heavily infected and they can provide advice about place to avoid other helpful measures include:-

1. Wear long-sleeved shirts and pants of medium-weight material in neutral colours that blend with the background environment. Tse-Tse flies are attracted to bright or dark colours and they can bite through light weight clothing.
2. Inspect vehicles before entering the flies are attracted to the motion and dust form moving vehicles.

3. Avoid bushes, the tse-tse fly is less active during the hottest part of the day but will bite if disturbed.
4. Use insect repellent permethrin-impregnated clothing and insect repellent have not been provide to be particularly effective against tse-tse flies, but they will prevent other insect bites that can cause illness.

   Control of African trypanosomiasis rests on two strategies; reducing the disease reservoir and controlling the tse-tse fly vector.

   Vector control is the primary strategy in use this is usually done with traps or screens.

   In combination with insecticides and odours that attract the files.

## Treatment

The doctors uses the antimony and arsenic compound for the trypanosomiasis Nitropurazone is used in cases resistant to arsenic.

## 2 BALANTIDIUM COLI :-

### Systematic position

Phylum - Protozoa
Class - Ciliata
Order - Spirotricha
Family - Balantididae
Genus - Balantidium
Species - B.Coli

### Geographical Distribution:

It is world-widely distributed. Because pigs are an animal reservoir, human infections occur more frequently in areas where pigs are raised other potential animal reservoirs include rodents and nonhuman primates.

### Morophology :-

1) Balantidium coil has two developmental stages

   i. A tropozoite stage.

   ii. A cyst stage.

2) In trophozoites, the two nuclei are visible.

3) The macronucleus is long and sausage-shaped and the spherical micronucleus is nested next to it, often hidden by the macronucleus.

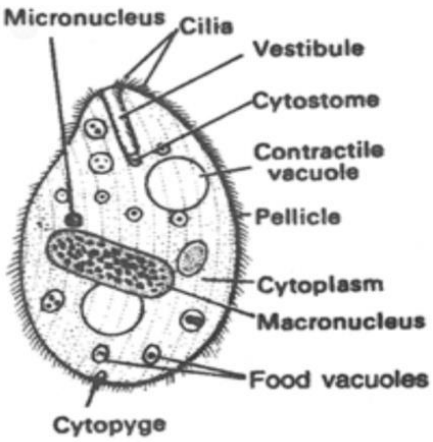
4) The opening, known as the peristome, at the pointed anterior end leads to the cystostome, or the mouth.

5) Cysts are smaller than trophozoits and are round and have a tough, heavy cyst wall made of one or two layers.

6) Usually only the macronucleus and sometimes cilia and contractile vacuoles are visible in the cyst.

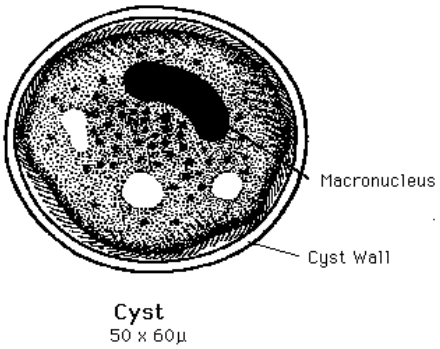
7) Living trophozoites and cysts are yellowish or greenish in colour.

Figure :- A) Trophozoites :-



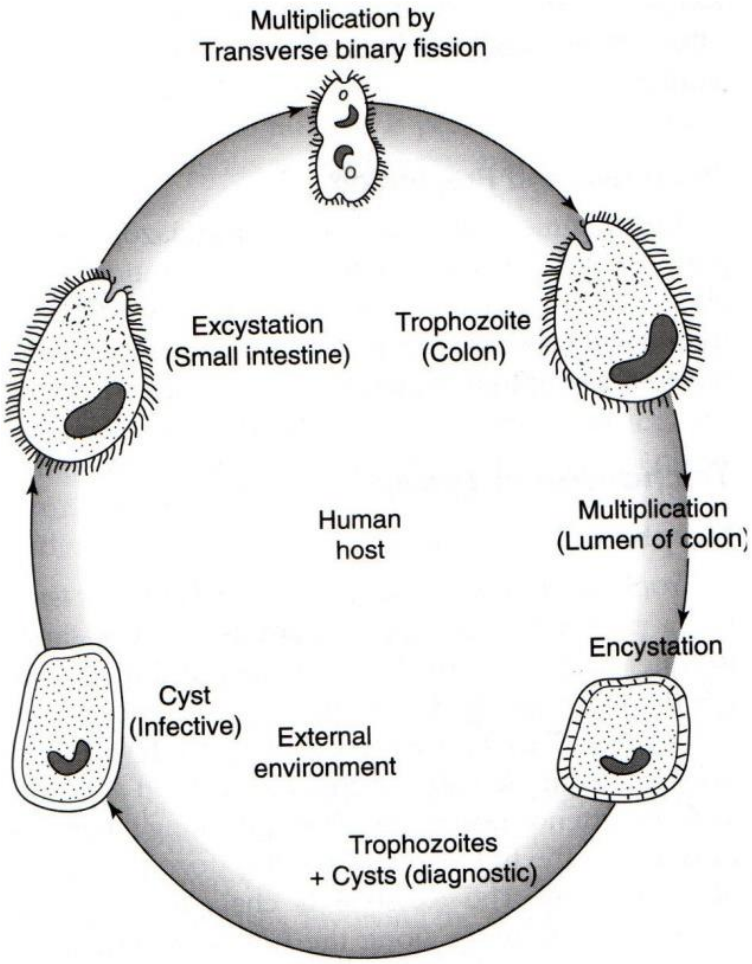

- Oval pointed at anterior end
- 50 – 130 μm long
- Covered in cilia
- Non-infective
- Reproductive by binary fission & conjugation
- Micronuclei & macronuclei

B) Cyst :-




- Spherical
- 40 – 60 μm across
- Covered with thick, hard cyst wall with cilia
- Infective
- Non-reproductive
- Macronuclei

Life-Cycle




1. Cysts are the parasite stage responsible for transmission of balantidiasis.
2. The host most often acquires the cyst through ingestion of contaminated food or water.
3. Following ingestion, excystation occurs in the small intestine and the trophozoites colonize the large intestine.
4. The trophozoites reside in the lumen of the large intestine of humans and animals, where they replicate by binary fission, during which conjugation may occur.
5. Trophozoites undergo encystations to produce infective cysts.
6. Some trophozoites invade the wall of the colon.
7. Particularly when if find unfavourable condition it also reproduce sexualy by conjugation during conjugation two trophozoite are include in a

exchange of nucleus material occur individuals separate, each egg. Conjugates a transfers binary fission to produce daughter form.

8. If the cystic form in the faeceal matter swallowed by another host infections starts. This B. Coli complete its life-cycle in a single host i.e. pig or man, hence it is called 'monogenetic cycle'.

## Pathogenicity :

Ciliate parasite causes dysentry called ciliate descentry the various symptoms are of balantidiasis are as follows :-

If affect with gastro intestine track causing various disorders of alimentary canal. Such as abdominal pain, weakness, constipation etc. due to invating of the parasite to the epithelial cells of intestine occilaration.

In this case the liquid faeces contains submucous, blood, mucous, neetrotic process, generally extend through the mucous & sub mucous part.

## Diagnosis :

Balantidiasis is an uncommon infection symptoms. If present, include diarrhea, dysentery and abdominal pain.

Balantidiasis should be considered if the patient works closely with pig or other livestock, lives in or has recently travelled to region with poor water sanitation or had contact with infected persons.

## Prophylaxis :

1) Avoid ingestion of material contaminated with animal faeces.
2) Prevention & control sanitary disposal of human and big faces.
3) Treatment of infected pigs.
4) Prevention of facial contamination of food and water.

## Treatment :

Drugs used for chemotheraphy are arisenical preparations and diwdohydroxy qulnoline oxytetracycline, has also been found to be an effective remedy emetin injection has been found to be of no value in balantidium injection.

## 2.2.3. *SARCOCYSTIS CRUZI*

### Systematic position :-

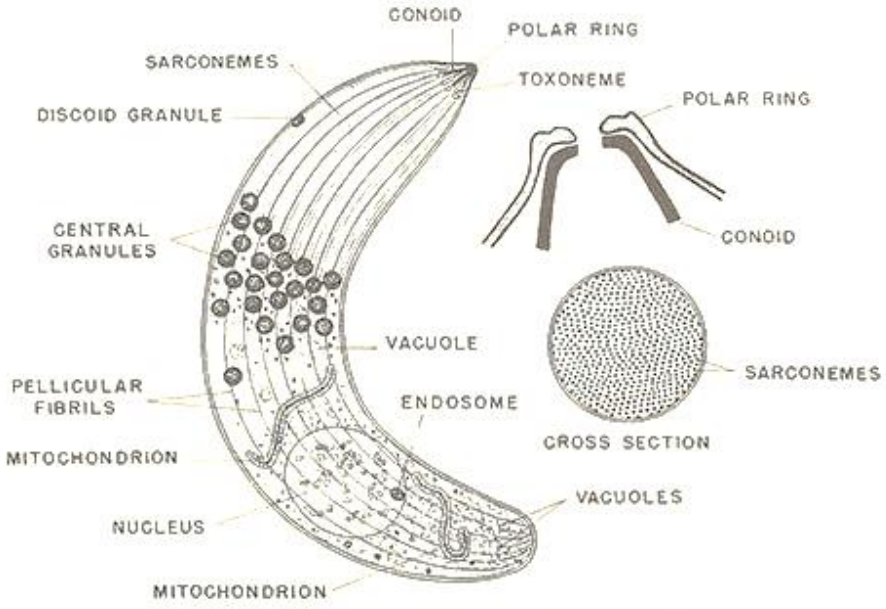
Phylum - Protozoa
Class - Sporozoa
Order - Eucoccidiorida
Family - Sarcocystidae
Genus - Sarcocystis
Species - *S. cruzi*

### Geographical Distribution :-

Sarcocystis infections occurs world wide but the frequency of infection is relatively low in humans.

**Morphology :-** There are three forms of sarcocystis –

(1) Oocyst (2) Sporocyst (3) Sarcocyst




TROPHOZOITE OF SARCOCYSTIS TENELLA

Oocyst and sporocyst develop in humans while sarcocyst is present in muscle of pig or cattle.

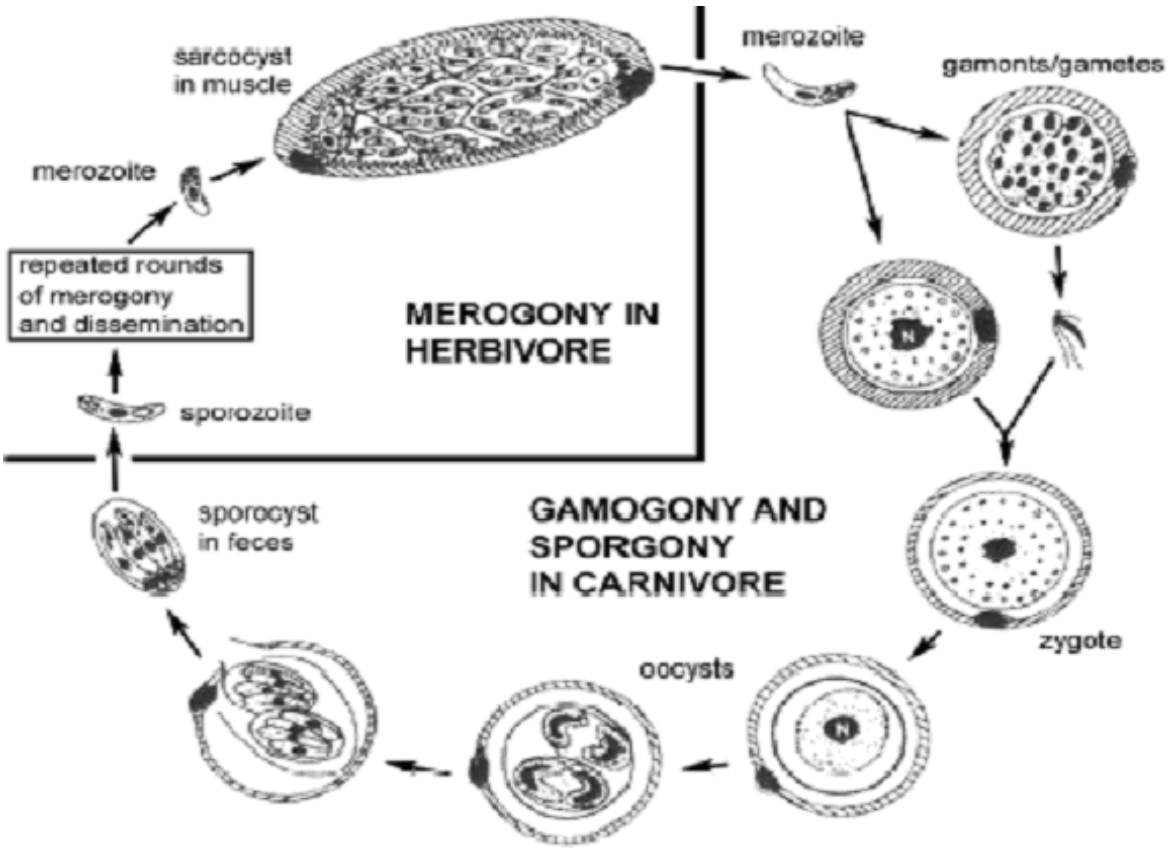
**Oocyst :** Oocyst is thin walled and colourless. Each Oocyst develops two sporocysts while four sparozoites are present in each sporocyst. It measure 13-9 μm in suihominis.

**Sporocyst :** Sporocyst is oval in shape and measure 8 to 10 μm in diameter. It is found in human faeces and is the infective form. Each sporocyst contains four sporozoites.

**Sarcocyst :** Sarcocysts of *s. hominis* and *s. suihominis* are found in cattle and pigs respectively.

These are present in the muscles of animal. These are also named as muscular cysts. These cysts are elongated and are found along the length of the muscle fibre. Each cyst is divided into many compartments which contain many bradyzoites Diaphragm, Oesophagus and Cardiac muscles are most frequently involved and contain sarcocysts.

Life Cycle



*S. hominis and S. suihominis* :

Life cycle is passed in two hosts definitive and intermediate. Definitive host is man while cattle and pigs are intermediate hosts for *S. hominis* and the *S. suihominis* respectively. Humans acquire infections by ingestion of raw or undercooked beef or pork containing sarcocysts. Bradyzoites are liberated from the sarcocyst and penetrate the intestinal mucosa and transform into microgamete (male) and macrogamete (female) fertilization occurs and the zygote. An Oocyst develops form the zygote subsequently, two sporocysts are formed in each oocyst. This oocyst is called sporulated oocyst. Each sporocyst contains four sporozoites and is infective to intermediate host. The sporulated oocysts are shed into the lumen of small intestine and sporocysts are released in the faces of definitive host.

The intermediate host becomes infected by ingestion of contaminated food or water containing sporocysts. Sporozoites are released from sporocyst in the small intestine. These sporozoites migrate to the blood stream and the produce schizonts in the vascular endothelium. Two generations of schizonts are formed merozoites are liberated from schizoints. The merozoites enter the skeletal and cardiac muscles and develop into sarcocysts containing numerous bradyzoites the raw of undercooked beef or pork. Sarcocysts is ingested by humans and life cycle is repeated.

*S. Lindemanni* :

Human (intermediate host) acquires infection by ingestion of food and drinks contaminated with sporocysts excreted in the faeces of cats, dogs or other carnivorous animals (definitive hosts). The life cycle of *S. lindemanni* is similar to that of *S. hominis* and *S. suihominis* with the difference that humans act as intermediate host instead of definitive host.

## Pathogenicity

*S. hominis* and *S. suihominis* cause intestinal sarcocystosis but *S. lindemanni* is a causative agent of muscular sarcocystosis. The intestinal sarcocystosis is usually asymptomatic but patient may develop nausea, pain abdomen and

diarirhae. The disease is self limiting. The muscular sarcocystosis is also generally asymptomatic but muscular pain, muscular weakness, myositis & pericarditis may be present.

Diagnosis :

Intestinal Sarcocystosis

Faeces examination – Characteristic sporocysts can be demonstrated in the faeces of human being. Two species of sarcocystis cannot be differentiated on the basis of sporocyst examination.

Muscular Sarcocystosis

Diagnosis can be made by demonstration of sarcocysts in the skeletal and cardiac muscle by biopsy or authopsy in human beings.

Prophylaxis

Intestinal sarcocystosis can be prevented by not eating raw or undercooked beef or pork. However, muscular sarcocystosis is prevented by avoidance of contamination of food & drink with faeces of cat, dog or other carnivorous animals.

Treatment

Current treatment are not entirely satisfactory. Amprolium (100 mg/kg sid for 30 days), fed prophylatically, reduced illness in cattle inoculated with S. cruzi.

## 2.2.4. *PLASMODIUM VIVAX* :-

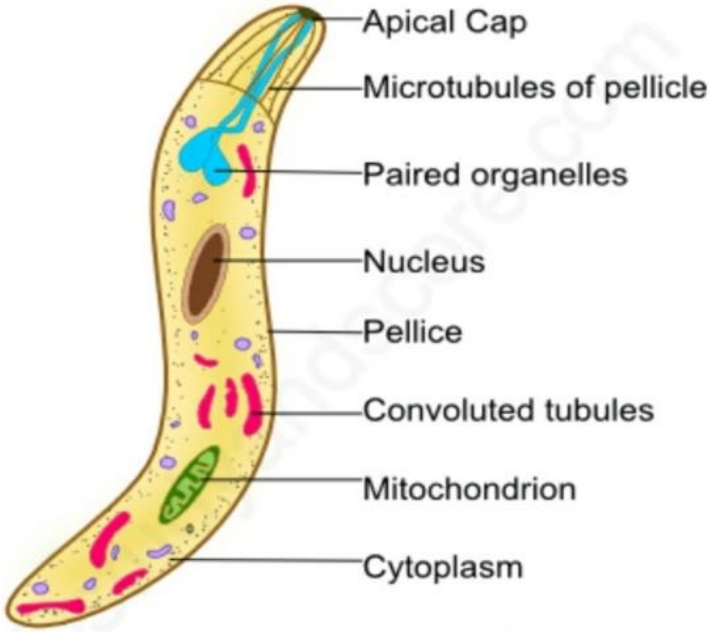
- Systematic position :-

Phylum - Protozoa
Class - Sporozoa
Order - Haemospororida
Family - Plasmodiidae
Genus - Plasmodium
Species - P. Vivax

Geographical Distribution : Malaria generally occurs in areas where environmental conditions allow parasite multiplication in the vector – P. malariae has wide global distribution being found in South America, Asia and

Africa, but it is less frequent than P. falciparum in terms of association with cases of infection.

- Morphology :-

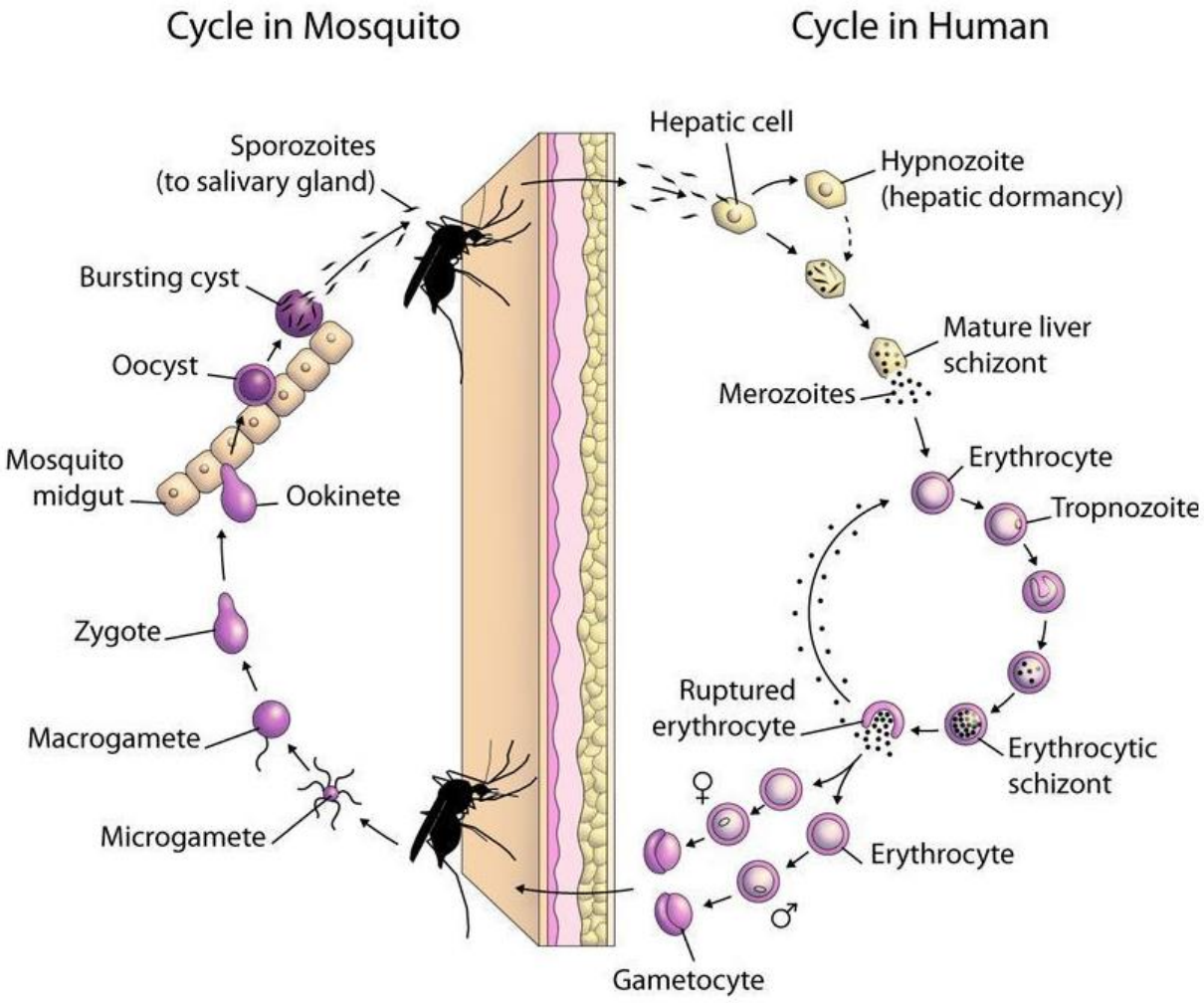


STRUCTURE OF SPOROZOITE

1) *Plasmodium vivax* causes benign tertian malaria, so called because the paioxysm of fever occurs every other day and the disease is rarely fatal. Plasmodium is an endoparasite which lives within the red blood corpuscles of man to whom it is transmitted by the bite of an infected female mosquito.
2) Thus the parasite has two hosts - man and Anopheles – and there is an alternation of on the hosts in its life cycle. Laveran discovered P. Vivax in 1890 in the blood of man but its connection with the insect – host and its mode of transmission was worked out in Calcutta by Ross in 1899. Plasmodium is the most important sporozoon which infects man.
3) It cause wide – spread havoc in tropical and subtropical regions, especially West Bengal, Bangladesh, Behar and Assam. There are three species of malarial parasite – P. Vivax, P. Malariae and P. falciparum. There life cycles closely resemble one another but the symptoms which they produce vary.

4) Thus P. Vivax is responsible for benign tertian malaria, the meaning of which has already been explained. P. malaria causes quartan malaria, so called because the outbreak of fever sets in every fourth day. P. falciparum produces malignant malaria in which the fever is irregular and the disease proves fatal unless the victim is promptly treated by antimalarial drugs. In untreated cases the parasites check up the capillaries of the brain.

Life Cycle :



Like all malaria parasites, P. Vivax has a complex life cycle. It infects a man.

Hosts :- Plasmodium completes its lifecycle in two hosts (digenetic) : man & female Anopheles mosquito.

1) Primary or definitive host :- Female Anopheles mosquito is the primary host of plasmodium in which it completes its sexual life cycle.

2) Secondary or Intermediate host :- Man is the secondary host of plasmodium in which it completes its asexual life cycle. The lifecycle of plasmodium can be divided into three phases.

1) Asexual schizogony.
2) Sexual gamogony.
3) Asexual sporogony.

## Asexual cycle of Plasmodium in Man :

Infective from of plasmodium is known as sporozoites. Sporozoites are 11-72 μ long slender uninucleated. Sickle-shaped structure present in the salivary glands of infected mosquito. When an gland of infected mosquito to female Anopheles mosquito bites a healthy man, a large number of sporozoites enter into the blood stream of man, within half an hour sporozoites enter the liver cells and undergo asexual multiplication called schizogony.

## Asexual Schizogony :

Schizogony is the asexual phase of reproduction of plasmodium it takes place in liver cells and RBC's of man schizogony can be divided into following phases –

a) Pre-erythrocytic schizogony.
b) Exo-erythrocytic schizolony.
c) Erthrocytic schizogony.
d) Post-erythrocytic schizogony.

a) Pre-erythrocytic Schizolony :

In the liver cells, sporozoites grow to form a large and spherical schizont. Schizont divides by multiple fission and forms a large number of cryptozoites. They may either pass into the blood circulation to start erythrocytic schizogony or enter fresh liver cells to start. Exo-erythrocytic schizogony. Pre-erythrocytic schizogony takes 7 days to complete.

b) Exo-erythrocytic schizogony :

After re-entering fresh liver cell each cryptozoites divides to form a large number of metacryptozoites similar to pre-erythrocytic schizogony meta-crypto zoites are two types – (1) smaller micro-metacryptozoites and (2) larger macro-metacryptozoites. It takes normaly 4 days to complete.

c) Erythrocytic Schizogony :

As stated above the erythrocytic schizogony begins when the RBC's of blood are attacked either by pre-erythrocytic cryptozoites or by exo-erythrocytic micro-metacryptozoites. It takes normally 8 to 12 days after above 2 phases. Stages of erythrocytic schizogony are.

i) Trophozoite State :- The merozoites after entering into the blood stream, feed on erythro-cytes, become rounded and modify into trophozoite.

ii) Signet Ring Stage :- As the merozoites grow a vacuole appears in the center and the nucleus is pushed to one side it gives a ring like appearance and known as signet ring stage.

The parasite ingests haemoglobin and decomposes it into protein and haematin. Protein is use as food whereas unused haematin forms toxic. Yellowish brown malarial pigment haemozoin.

iii) Amoeboid Stage :- As the signet ring parasite grows vacuole disappears and the parasite becomes amoeboid in appearance thrusting out pseudopodial processes. This stage is called amoeboid stage. At this stage RBC develops numerous granules the schuffner's granules.

iv) Schizont Stage :- Parasite grows in size becomes rounded and almost completely fills the RBC called schizont.

v) Rosette Stage :- The nucleus of schizont divides by multiple fission to form 6 to 24 daughter nuclei. These nucles arrange at the periphery, while the toxic haemozoin granules accumulate at the center of RBC it appears as a flower rose, so called rosette stage.

d) Post-erythrocytic Schizogony :

Sometimes, some merozoites produced in erythrocytic schizogony reach the liver cells and undergo schizogony development in liver cells. This is called post-erythrocytic schizogony.

Sexual Cycle of Plasmodium in Man :-

2) Sexual Gamogony :-

Formulation of gametocytes :- After many generations in about 4-5 is the blood some merozoites increase in size to form two types of gameteocytes; larger macro (9-10μ) less numerous & contain large nucleus, macro gametocytes are larger (10-12μ) more numerous and contain smaller nucleus.

- Sexual Cycle of Plasmodium Mosquito :- When a female Anopheles suck the blood of a malaria patient the gametocytes reach the stomach of mosquitoes and formation of gametes takes places.

a) Gametogenesis :- (gemetogony)

Process of formulation of gametes (male & female gametes).

i) Formation of male gametes :- The nucleus of microgametocyte divides to form 6-8 daughter nuclei. The cytoplasm gives out same number of flagella like projections and daughter nuclei enter in each projection. These projections separate from the cytoplasm & form 6-8 haploid microgamete or male gametes. This process of formation of microgamete is called flagellation.

ii) Formation of female gamete :- The mega gametocyte undergoes some reorganization to form a single haploid male gamete or female gamete which is ready for fertilization.

b) **Fertilization :-** The male gamete enters the female gamete through the fertilization cone formed at female gamete through the fertilization cone formed at female gamete & form diploid zygote or synkaryon fusion is anisogamous.

c) **Ookinete Stage :-** The zygote remains inactive for sometimes & then elongates into a worm like ookinete or vermicale which is motile. The ookinete penetrates the stomach wall and comes to lie below its outer epithelial layer.

d) **Oocyst Stage :-** The Ookinete gets enclosed in a cyst. The encysted zygote is called Oocyst. The Oocyst absorbs nourishment & grows in size.

4) **Asexual Sporogony :**

The nucleus of Oocyst divides repeatedly to form a large number of haploid daughter nucles. At the some time the cytoplasm develops vacuoles and gives numerous cytoplasonic masses. The daughter nuclei pass into each cytoplasmic mass and develop into slender sickle-shaped sporozoites are formed in each Oocyst. This phase of asexual multiplication is known as sporogony.

Lastly, the Oocyst brusts and sporozoites are liberated into the haemolymph of the mosquito. They spread throughout the haemolymph & eventually reach the salivary glands & enter the duct of the hypopharyx. The mosquito is now becomes infective & sporozoites get inculated or injected the human blood when the mosquito bites the cycle is repeated.

**Pathogenicity :-**

5) Infection causes intermittent fever – Malaria.

6) Each of the 4 species causes & characteristic fever.

| | | |
|---|---|---|
| P. Vivax | - | Benign tertian / Vivax malaria. |
| P. falciparum | - | Malignant tertian / falciparum Malaria, black water fever. |
| P. malariae | - | Quartan malaria |
| P. ovale | - | Tertian / ovale malaria |

7) Paroxysm (attack of malaria)

Mechanism – liberation of merozoites & malarial pigment RBC debris into the blood stream.

## Diagnosis :

The diagnosis and treatment of *plasmodium vivax* malaria differs from that plasmodium falciparum malaria in fundamentally important ways. This artiole reviews the guiding principles, practices & evidence under pinning the diagnosis & treatment of P. Vivax malaria.

The accurate diagnosis of Vivax malaria in an acutely ill patient seeking routine care requires microscopy examination of a Giemsa – stained blood smear or use of an immune chromatography in P. vivax.

## Prophylaxis:

Indication.

Duration :- 1-2 weeks before to 4 weeks after returning from endemic area.

## Drug regimens :

- Chloroquine sensitive malaria – 300 mg / week.
- Chloroquine resistant malaria;
    1) Mefloquine 250 mg once a week.
    2) Doxycycline 100 mg daily.
    3) Atovaquone + proguanil daily.

**Treatment :-** Chloroquine is used for treatment of acute malaria. However, resistance to this drug, malaria in P. falciparum is reported widely.

The drugs resistance in P. falciparum is mainly due to.

1) Use of inadequate drug does by non-immune & semi-immune persons for treatment.
2) Use of low doses of drugs for prophylaxis.
3) When the insufficient drug is used to kill the malarial parasites the mutants survive & multiply.

## 5) EIMERIA TENELLA :

### Systematic position :

Phylum - Protozoa
Class - Conoidasida
Order - Eucoccidiorida
Family - Eimeriidae
Genus - Eimeria
Species - E. tenella

### Geographical Distribution :

It is more common in distribution world poultry industries exist, it is a more common in India.

### Morphology :-

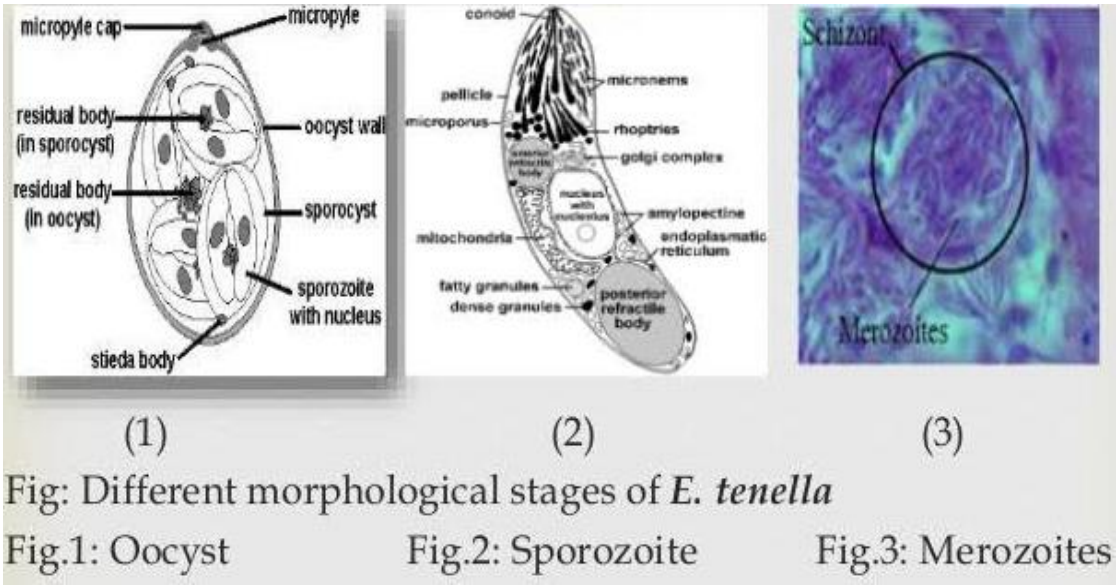



Fig: Different morphological stages of *E. tenella*

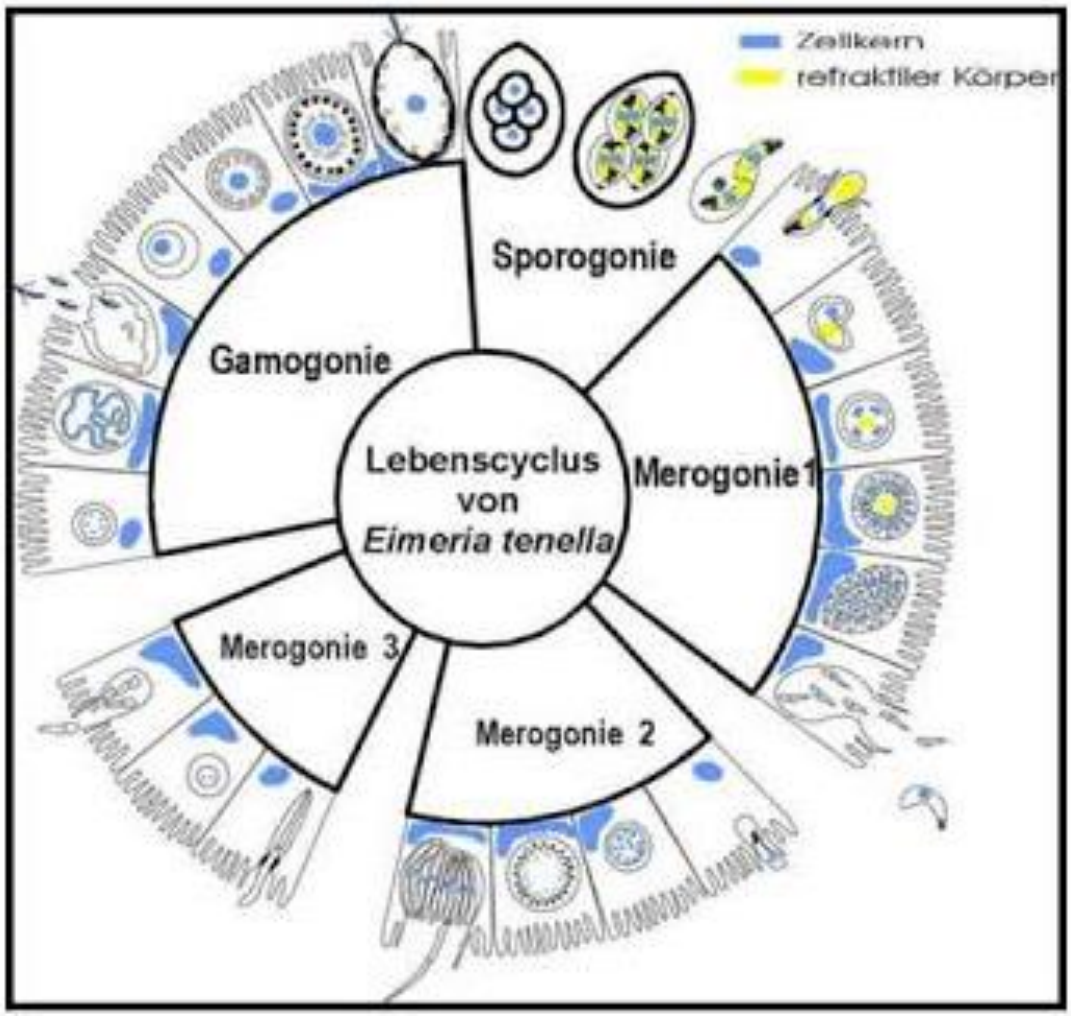
Fig.1: Oocyst Fig.2: Sporozoite Fig.3: Merozoites

The parasite Eimeria tenella occurring in many kinds of cell of vertebrates and in instestinal epithelium of invertebrates E. tenella causes great economical loss in domestic animal serious out breaks of infection known as coccidiosis occur in poultry. This disease is more common in the chickens.

The infective form of parasite is called as sporozoites. These are elongated slightly curved microscopic unicellular organism they have one end point & other end point is blunt.

At the end of 1st and 2nd asexual cycles periform organisms are produced. These are called as merozoites. They are spindle shaped. These are measuring about 2-4 microns in length & 1 to 1.5 microns in width. The second generation merozoites measures 16 microns in length & 2 microns in width.

Life-Cycle :




It is a monogenetic life cycle involves only one host, chicken to complete it's cycle is called monogenetic its cycle is completed in two phases.

1) Asexual cycle, schizogony.
2) Sexual cycle involving gametogony or sporogony.

1) Asexual Cycle :- (Schizogony)

This cycle has again two phases.

i) First generation schizogony.

ii) Second generation schizogony.

It starts by consuming infective forms called sporozoites. The Oocytes containing sporozoites or swallowed by the sporozoites liberate & infective epithelial cells of host the sporozoites grow in epithelial cells & multiply by schizogony.

i) Sporozoites :- The sporozoite is first infective intracellular parasite stage. It is an elongated slightly curved microscopic unicellular body with one end pointed and other end is blunt it's body is curved by non-flexible external sporozoites enter into epithelial cells, convert into trophozoite.

- Trophozoites :- The sporozoite in epithelial cell of the host observed food it grows inside & become bean shaped it contains esosinaphil globules. The troporoites observes pregasted food from the cytoplasm of the host cell. It increases in size it's nucleus undergoes repeated division & this method is called as schizont.

Schizont :- The trophozoites forms into schizont the repeat mitotic nuclear division it result in the formation of multinucleated called schizont. It is a oval shaped. With loculated cytoplasm the outer margin with small nucleoli, like also contain esosinophil globule the schizont give rise to organism called merozoites.

- First generation merozoites :- Within the schizont nuclei with cytoplasm masses are placed on the peripheral margin. This masses are converted into merozoites a single schizont may give rise to 900 merozoites this are called as first generation merozoites. Each merozoite is a small measuring about 2-4 micron in length and 1 to 1.5 micron in width it's one end is pointed while the other end is rounded it's nucleus is vascular, present in the center.

- Second generation schizogony :- The first generation merozoites attack new fresh epithelial cells of host this come and live in cytoplasm and grows into trophozoites.

**Trophozoites :-** This trophozoites are similar to first generation but it is without esosinophil globules it feed up on the host cytoplasm its nucleus undergo repeated mitatid division from a multinucleat schizont.

**Schizont :-** The second generation schizont this all together different from the first generation. It is larger in size and its nuclei are scattered throughout its cytoplasm is daughter nuclei the nucleated cytoplasmic masses gives rise to second generation merozoites.

**Second generation merozoites :-** The second generation merozoites is also elongated and peripheral form with rounded and other end is blunt they are bigger than first generation merozoites. It measuring about is micron length & 2-4 width. There number in single schizont about 250 the host epithelial cells brust along the schizont it results in the liberation of second generation merozoites.

2) **Sexual Cycle :-** Some merozoites in the host epithelial cells become male or microgameto-cytes while some become female or macro-gametocytes. Microgametes is small and oval, measuring about 5.5 to 18 microns in length the macrogameto cytes is also oval and larger and measuring 8-25 microns length. This process is called as formation of gametocytes. The microgametocytes repeated nuclear division & give rise to neumerous, biflagellate male cells called microgamets.

**Fertilization :-** The microgametes come out from the host cell & attack new cells containing macrogameto-cytes. The micro gametocytes with macrogametes. Then the fertilization take place inside the cell after fertilization its result zygote is formed it is diploid. The zygote know secretes and forms into thick cyst wall around it self its cyst wall is double with a thicker outer end inner delicates the outer thick layer is gunar tunned protein while the inner layer is of lipo-protein layer.

**Oocysts :-** It is oval in shape and measuring about 20-26 $\mu$ by 16.5 – 23 $\mu$. Thiocysts fall in the quity of the intestine by the along with the faeces of bud. Generally Oocyst passes outside on the $7^{th}$ or $8^{th}$ days of infection the Oocysts remains active for a longer period anaerobic conditions.

- **Pathogenicity :-** Eimeria Tenella causes a foetal disease of sickness called sickle coccididiasis. In this diseases there is a distribution of sick epithelium and worm.

There is a survivor hamarage, the symptoms include blooding droppings pales face and enlarge sick a distended with blood or with grey like series. The infected bird show release droping wings, suffeled feathers. This disease is more survivor in the infact than the young and old one.

- **Diagnosis :-** The objective of this study was to identify and characterize species of Eimeria in broiler chickens using traditional morphological and pathological plus molecular diagnostic methodologies.

The frequencies of the other species were Eimeria mitis & Eimeria necatrix (93.3%) Eimeria tenella (76.7%), Eimeria acervulina (56.7%)

DNA amplification had detection sensitivity for Eimeria species in the field samples of at least 20 Oocysts.

The implementation of DNA amplification as a routine diagnostic technique in a aviaries can assist Eimeria population.

- **Prophylaxis :-**

The following are the control measure to control foetal disease called sick coccidiosis.

i) The poultry and surrounding area kept regularly cleaning and removing water the poultry houses must be clean.

ii) The Oocysts must be destroyed by use of $NH_3$.

iii) The infected birds are kept a side and treated immediately with sulphur drugs like sulphar mesathin & sulphar quinaoxline.

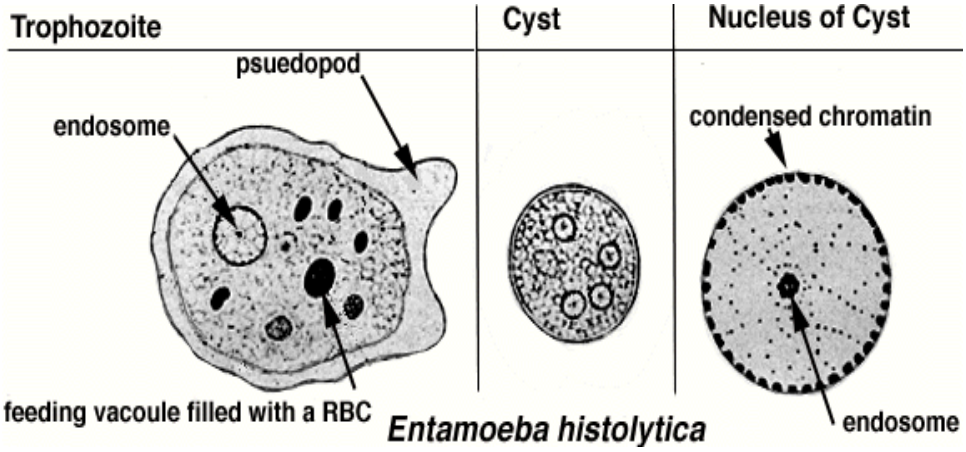
- **Treatment :-** Lonophorous antibiotics have been popularly used in the treatment of avian coccidiosis tissue residue of these antibiotics may be found in poultry, we have sought safe alternative anticoccidial herbal materials for the control of avian coccidiosis.

## 6) ENTAMOEBA HISTOLYTICA :-

- **Systematic position :-**

Phylum - Protozoa
Class - Rhizopoda
Order - Lobosa
Genus - Entamoeba
Species - E. histolytica

- **Geographical Distribution :-** It is world widely distributed more common in tropics and sub-tropics than in the temperate zone.
- **Morphology :-**




The parasite causing diarrhea, dysentery and liver abscess in man.

Lambl (1859) first discovered the parasite Losch (1875) proved it's pathogenic nature. Entos = within, Amoeba = change, Histos = tissue, Lysis = Dissolved

It morphology present in three forms :-

(1) Trophozoite (2) Pre-cystic phase (3) Cystic phase

### 1) Trophozoite :-

**Shape :-** Not fixed because of constantly changing position.

**Size :-** Ranges from 18 to 40 μm. avg. 20-30 μm.

**Cytoplasm :-** Divisible into two portion a clear translucent ectoplasm and a granular endoplasm.

**Nucleus :-** Spherical in shape and varying in size from 4 to 6 μm.

### 2) Pre-cystic stage :-

It is smaller in size, varying from 10 to 20 μm. It is round or slightly avoid with a blunt pseudopodium projecting from the periphery. The endoplasm is free of RBC's and other food particles.

### 3) Cystic phase :-

During encystment the parasite becomes rounded and it surrounded by a highly refractile membrane, called cyst wall.

A mature cyst is quadrinucleate spherical body, it's cytoplasm is clear and hyaline. The size varies in cyst the small range is 6 to 9 μm & large-range 12-15 μm.

### Life-cycle :

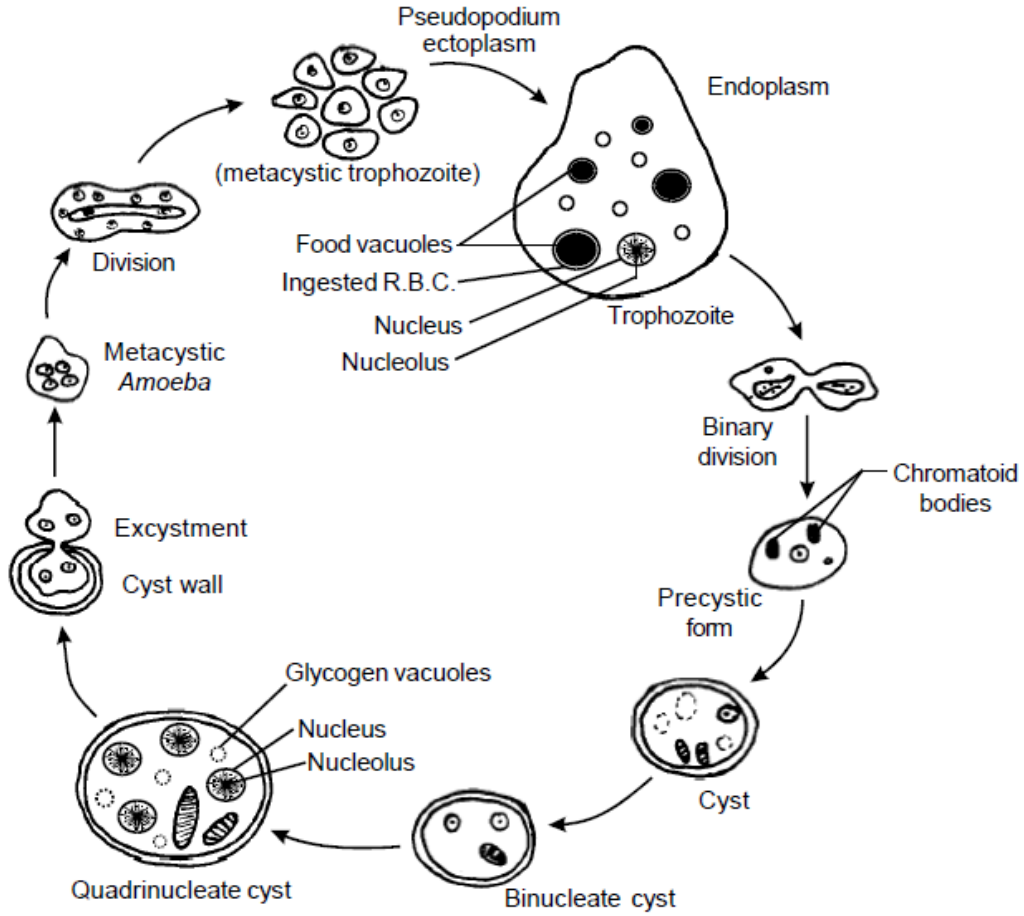



**Fig. *Entamoeba histolytica* and its life cycle**

The life cycle of Entamoeba histolytica is monogenic lie. It is completed in a single host only. The parasite are transmitted from one host to another host in encysted stage.

The pre-cystic form undergo encystment but before encystment, the parasite round up eliminate food vacuoles and accumulate considerable amount of food materials in the form of glycogen & black rod like chromatid granules. The nucleus of cyst divides twice so that each cyst is infective to new host. Encysted forms pass out with the faecal matter of the host.

The infective cyst remain viable for a long time out side the human intestine. Infection of human host takes place by spoiling the infective cysts with contaminated food and drinks.

In the process of excystation a single tetranucleate amoeba emerges from a cyst through a minute pore in the cyst wall and these produce new generation of the trophozoite.

## Pathogenicity :-

In man the incubation period varies but generally four or five days.

The term amoebasic is used cleanically to denote all those conditions such as proceduced in the human host by infection with E. histolytica at different which areas of it's invasion. The term amoebic dysentery signifies a condition in which the infection is confined to the intestine canal and is characterized by the passage of blood and mucus in the stool. The form "amoebic dysentry" is not a synonym of 'amoebiosis' it gives full picture of the manifestation of intestinal amoebiosis.

## Diagnosis :-

Diagnosis is confirmed by micro-scopic examination for trophozoites or cysts in fresh or suitable preserved faecal specimens, smears of aspirates or sceapings obtained by proctoscopy and aspirates of abscesses or other tissue specimen.

A blood test is also available bat is only recommended when a healthcare provider believes the infection may have spread beyond the intestine to some other organ of the body, such as the liver.

Stool antigen detection and PCR are available for diagnosis and are more sensitive and specific than microscopy.

## Prophylaxis :-

1) Adequate water purification. Low levels ofd chlorine does not kill cysts.
2) Boiling of drinking water.
3) Treating vegetables & fruits with strong detergents soap & then soaking in acetic acid for 5-10 minutes.
4) Proper waste disposal.
5) Immunization under trial.
6) Detection of isolation of carriers.

## Treatment :-

In the intestinal wall liver and other metastatic infections the drugs emetine & dehydroematine are used.

The most common treatment for giardiasis is –

1) Metronidazole (flagy) for 5-10 days.

It eradicates giardia in more than 85% of cases.

2) Furazolidone (furoxone) for 7-10 days.
3) Quinacrine is also very effective for treating giardiasis.

- Combination therapy also may be effective.
- Quinacrine & metronidazole.
  Tinidazole, secnidazole & Albendzole.

## 7) ENTAMOEBA COLI :-

- **Systematic position :-**

Phylum - Protozoa
Class - Rhizopoda
Order - Lobosa
Family - Entamoebidae
Genus - Entamoeba
Species - E. Coli

- **Geographical Distribution :-**

It is world – widely distributed it lives in the lumen of large intestine. It is non-pathogenic.

- **Morphology :-**

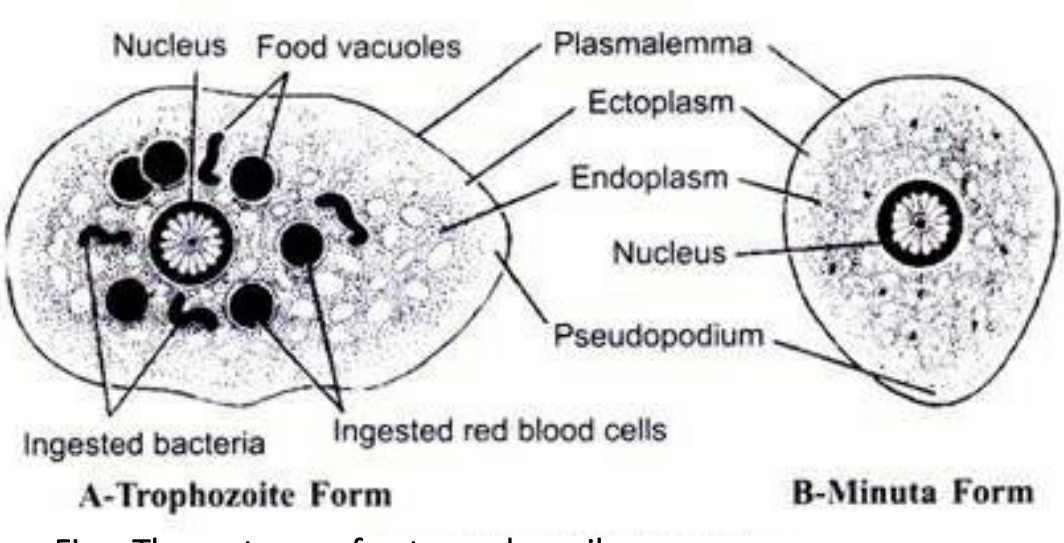



Fig – Three stages of entamoeba coil

(1) Trophozoites (2) pre-cystic form (3) to (6) cyst with one to eight nuclei

There are mainly two stage in the life-cycle with transitory stage of pre-cystic form –

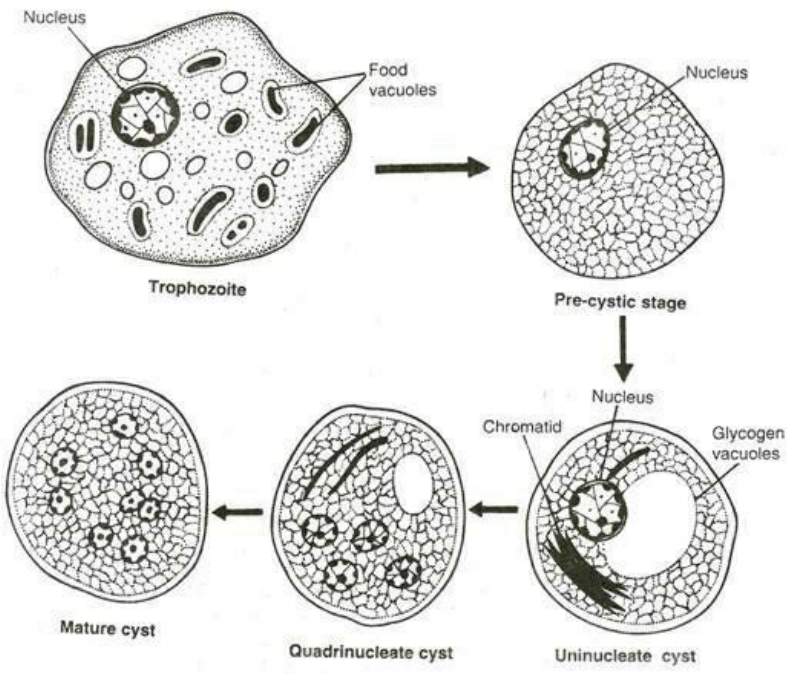
(1) Trophozoite (2) Cyst

**i) Trophozite :-** It is one of the largest amoeba accuring in the human colon and measures 20-40 μm. in diameter. It is sluggishly motile. The

cytoplasm is not clearly defined. The opaque granular endoplasm is packed with food vacuoles consisting of bacteria and other substance but never. RBC's because it is richer in chromatin the nucleus is visible in unstained preparation.

ii) Cyst :- It begins with a single nucleus & repeated nuclear divisions, on octonucleate cyst is formed. It is a rounded body measuring 15-20 μm. in diameter. In binucleate stage there is a large glycogen mass. Neither the glycogen mass nor chromatid bodies are to be found in nature cyst.

- Life Cycle :-




E Coli passes it's life cycle only in one host (man) There are mainly two phases of development trophozoite & cyst with a transitory stage of pre-cystic form.

The mature quadrinucleate cysts are the infective forms of the parasite. When these cysts are swallowed along with the contaminated food and drink by a suspectible person, they capable of further development inside his gut. The fully developed cysts, thus gaining entrance into the alimentary canal, pass unaltered through the stomach.

The cyst-wall is resistant to the action of gastric juice but is digested by the action of trypsin in the intestine. The excystation occurs when the cyst reaches the caecum or lower part of the ileum (neutral or slightly alkaline) medium). During the process cytoplasmic body, the cytoplasmic body retracts and 100 sens itself from the cyst wall. Vigorous amoeboid movement cause a rent to appear in the cyst wall through which at first a small mass of cytoplasm and then ultimately the whole body comes out. Each cyst liberates a single amoeba with four nuclei, a tetranucleate amoeba which eventually forms eight amoebulae (metacystic trophozoite) by the division of nuclei with successive fission of cytoplasm. The young amoebulae being actively motile, invade the tissue and ultimately lodge in the mucous fissue of the large gut, their normal habitat. Here, they grow & multiply by binary fission.

### Pathogenicity :-

Exist as a harmless commensal.

### Diagnosis :-

As E. coli is more commonly found in the dysenteric stoll.

### Prophylaxis :-

Sanitation is essential prevention is mostly depends on a personal hygin & sanitary disposal of human excreta.

### Treatment :-

1) There is generally no need to treat for E. coli due to the rarity of this parasite becoming infections.
2) In one exceptional situation, E coli was found to be infections.
3) Metronidazole & tinidazole.
4) Fluid & electrolyte replacement.

## 8) GIARDIA INTESTINALIS :- (Lamblia)

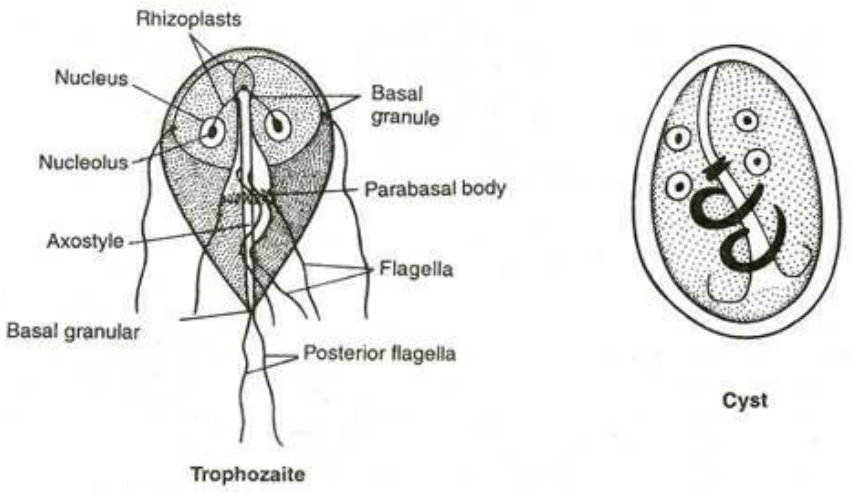
### Systematic position :-

| | | |
|---|---|---|
| Phylum | - | Protozoa |
| Class | - | Zooflagellate |
| Order | - | Diplomonadida |
| Family | - | Hexamitidae |
| Genus | - | Girdia |
| Species | - | E. Lamblia |

### Geographical Distribution :-

World wide, more prevalent in warmer climates and in children, infection occur in areas of low, sanitation standards & daycare centres.

### Morphology :-




G. Lamblia has two morphological stages; (1) Trophozoites (2) Cyst

### i) Trophozoites :-

The Trophozoites is pear shaped, with a broad enterior and much attenuated posterior.

It is 10-12 μm. long and 5-7 μm. wide, bilaterally, symmetrical and has two nuclei.

It is also relatively flattened, with a large sucking disk on the anterior ventral side, which serves as the parasite's method of attachment to the mucosa of the host.

The Trophozoite also has two median bodies and four pairs of flagella (anterior, caudal, posterior and ventral).

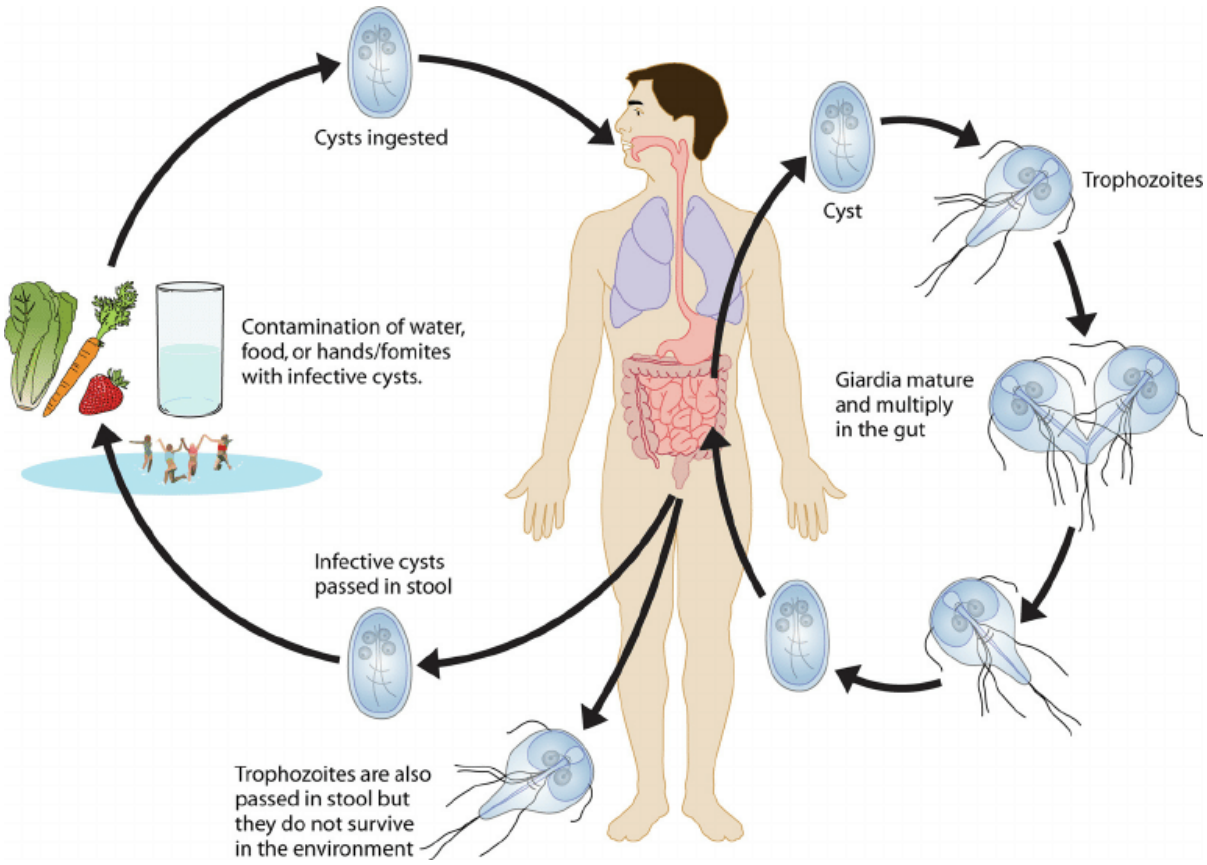
ii) Cyst :-

The G. Lamblia cyst is egg. Shaped, and measure 8-14 μm. by 7-14 μm.

After encystations, each organelle duplicates, so each cyst contains four nucles, four median bodies, eight parts of flagella-although these orgenelles are not arranged in any clear pattern.

Upon excystation, each cyst produces two trophozoites.

Life-Cycle :-



The life cycle begins with a non-infective cyst being excreted with the faces of an infected individual. The cyst stage can survive well in a variety of environmental conditions and even the acidity of the stomach therefore allowing it to pass through to the small intestine where it can replicate & cause clinical illness.

Cysts are then formed and passed through the faces to complete its life cycle and spread to other hosts. The cyst is hardy providing protection from various degrees of heat and cold, desiccation and infection from other organism.

Once ingested by a host, the trophozoite emerges to an active state of feeding and motility. After the feeding stage, the trophozoite undergoes asexual replication through longitudinal binary fission. The resulting trophozoites and cysts then pass through the digestive system in the faces. While the trophozoites may be found in the faces, only the cysts are capable of surviving outside of the host.

Distinguishing features of the trophozoites are large karyosomes and lack of peripheral chromatin, giving the two nuclei a halo appearance cysts are distinguished by a retracted cytoplasm. The protozoan lacks mitochondria, although the discovery of the presence of mitochondrial remnants in one recent study indicate that Giardia is not primitively a mitochondrial & that it has retained a functional organelle drived from the original mitochordrial endosymbiont this organelle is now termed a mitosome.

- Pathogenicity :-

1) Tightly adheres to intestinal epithelium.
2) Causes abnormalities of villous architecture by apoptosis.
3) Increased lymphatic infiltration of lamina propria.
4) Suction force.
5) Variant specific surface proteins (VSSP)
6) Virulence & infectivity.
7) Protection from proteases in intestine.

- Diagnosis :-

1) Stool examination (formed, loose)
   - Wet mount.

- Concentration.

2) Repeated samplings may be necessary.
3) Sample of duodenal fluid (e.g. Enterotest)
4) Serological Examination.
5) Antibody response.
6) Antigen detection.

- Prophylaxis :-

1) Adequate water purification.
2) Boiling of drinking water.
3) Treating vegetables & fruits with strong detergent soap & then soaking in acetic acid for 5-10 minutes.
4) Proper waste disposal.
5) Immunization.

- Treatment :-

1) Metronidazole & Tinidazole.
2) Fluid & electrolyte replacement.
3) Drainage of larger hepatic abcess.

CT guided percutaneous catheter drainage is useful both in resistance cases and perforation.

## 9) TRICHOMONAS VAGINALIS :-

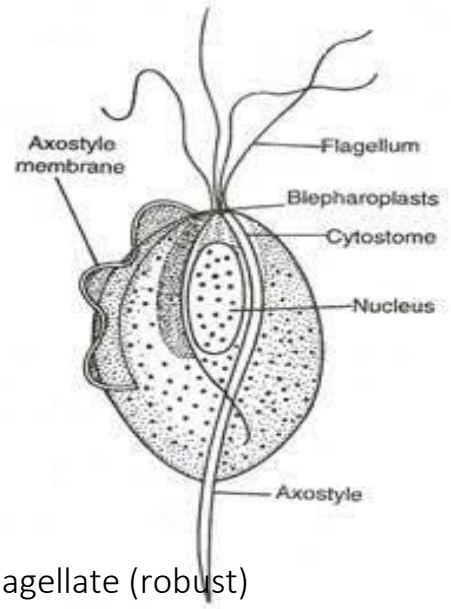
- **Systematic position :-**

Phylum - Protozoa
Class - Parabasalia
Order - Trichomonadida
Family - Trichomonadidae
Genus - Trichomonas
Species - T. Vaginalis

- **Geographical Distribution :-**

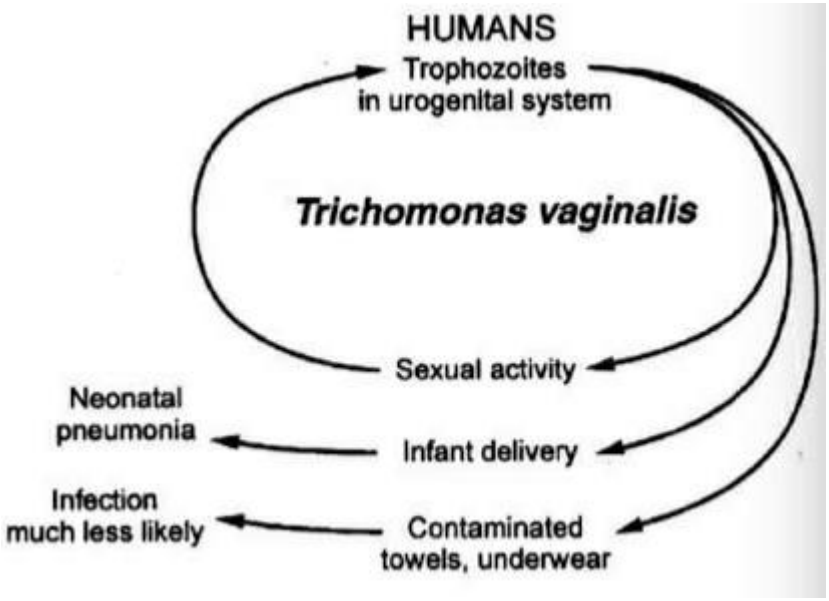
Worldwide, higher prevalence among persons with multiple sexual partners or other venereal diseases.

- **Morphology :-**




1) It is colourles pyriform flagellate (robust)
2) Size is 17 x 10 μ.
3) Anterior cleft – like cytostome.
4) One vesicular nucleus.
5) Four flagellae directed anteriorly.
6) Another one attach to 1/3 of the body of the parasite – undulating membrane.
7) The $6^{th}$ flagellum passes through the body as thin axostyle & projects out posteriorly.
8) A thick rod called parabasal body present between axostyle and the undulating membrane.

- Life-Cycle :-




1) Trichomonads have the simplest kind of protozoan life cycle, in which the organism occurs only as a trophozoite (No cyst).

2) It passes its life cycle in only one host, hamans, Trophoroites stage is the infective stage and is transmitted primarily via sexual contact.

3) It is occasionally found in virginal females / males and newborn wash cloths, clothes and towels.

4) It multiplies by longitudinal binary fission.

5) Reproduction of the parasites occurs every 8-12 hours.

Life cycle at a glance :-

Life cycle stage → only Trophozoites No cyst

Infective stage → Trophozoites

Pathogenic stage → Trophozoite

Route of infection → sexual & occassionaly via fomites

Site of Localization → vagina in women, prostate & seminal. Vesicles in men & the urethra in both sexes.

- Pathogenicity :-

Kolpitis, Urethritis.

- Diagnosis :-

1) Physical Exam : Nearly impossible.

2) Laboratory – microscopic observation of discharge very low sensitively.

3) Wright or giemsa stain.

- Prophylaxis :-

1) Because of the frequent role of asymptomatic men in spreading trichomoniasis, control of this infection necessitates examination and if necessary treatment of male sex partners.

2) Avoidance of sexual intercourse and the use of condoms are effective ways to prevent transmission.

3) Treatment of both sexual partners at the same time is recommended to prevent “ping pong” reinfection.

- Treatment :-

1) The treatment of choice is metronidazole, except in the first trimester of preganancy, when clotrimazole is used typically to 14 days, a large single dose of 2 gm by mouth is as effective as longer term treatment of 500 mg twice daily for 7 days. The 2 gm metronidazole dose has a 90-95% cure rate and good compliance since it is an all at once dose other nitroimazoles such as nimorazole or ornidazole are also effective.

2) It has also been suggested that the intravaginal application of the spermicide, nonoxynol I may have some effect against metronidazole resistant Trichomonas.

## 10) TRITRICHOMONAS FOETUS :-

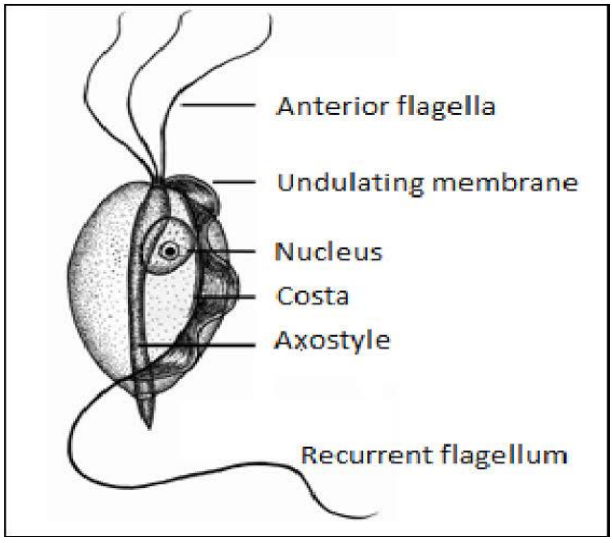
- **Systematic position :-**

Phylum - Protozoa
Class - Parabasalia
Order - Tritrichomonadida
Family - Tritrichomonadidae
Genus - Tritrichomonas
Species - T. Foetus

- **Geographical Distribution :-**

It was first observed by Redrawn in "1913". It is cosmopolitian in distribution but they are commonly found in tropical area generally the parasites occurs in the urinogenital system of cattles, sheeps, horse, etc. It causes uterine and pineal disorders causing inflammation and absess. Transmission or infection is direct through intercourse.

- **Morphology :-**




Trichomonus foetus is 10 to 15 microns length pear on oval shaped. It has three swimming flagella with an undulating membrane formed by the trailing flagellum. It helps in locomotion. The membrane or structure is called as costa or axostyle.

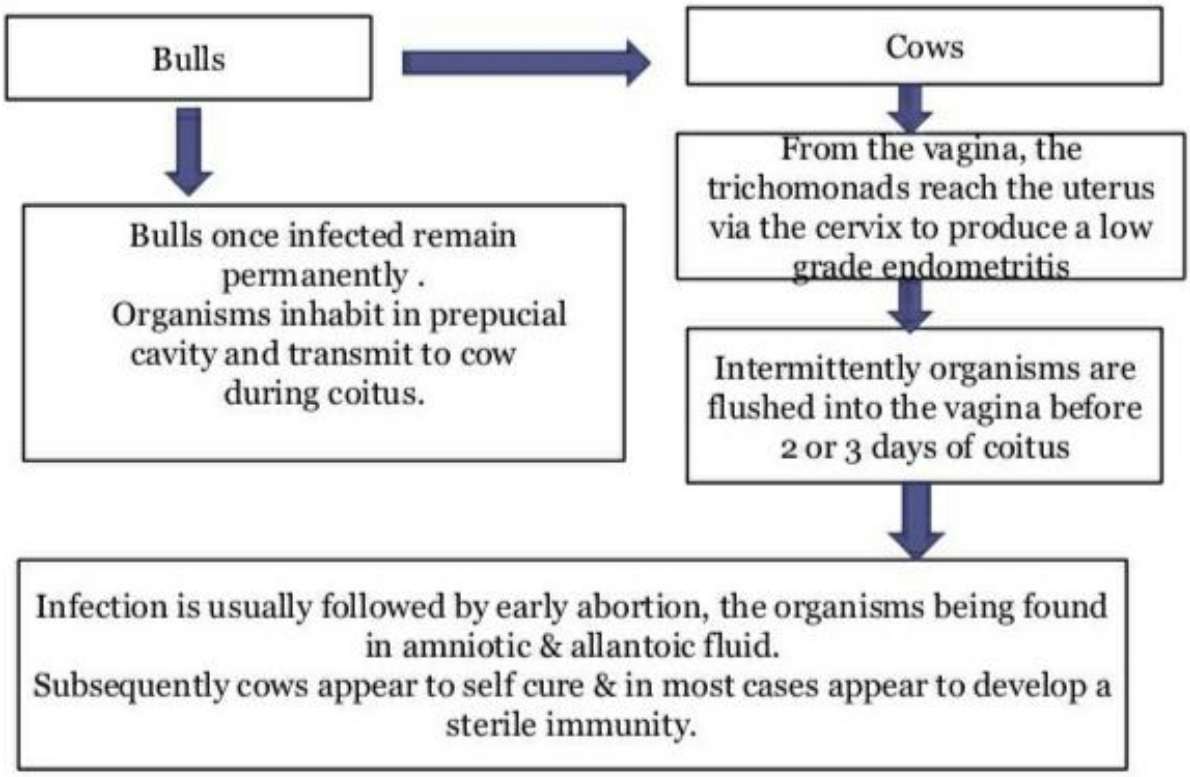
The Axostyle runs entire length of body & posteriorly tapering to form a spike. The whole body is filled with cytoplasm covered by pellicle which can be distinguished into ectoplasm & endoplasm in the cytoplasm. In the body of foetus metachromatic granules are present along the sides of axostyle. There is a single oval nucleus is situated & there is a cleft like depression called as cytozome lying the side.

- Life-Cycle :-

The life cycle of T. foetus in cats is direct, via the faecal oral route, Trophozoites are ingested from fresh faecal contamination, either from the coat.

It is unknown if this is a genetic predisposition or if these breeds are more commonly kept in high risk environments.

1) Trichomonas foetus, note three anterior flagellae & recurrent flagellum…… as fas as is known trichomonas foetus has a single life cycle stage.
2) Trichomonus foetus is a venereal disease in cattle causing early embryonic death & abortion.
3) The protozoa resisdes on the surface & in the lumen of the female reproductive fract & in the crupt of penile epithelial cells. Transmission is achieved from infected individuals during mating direct contact.
4) Tritrichomonas foetus is an obligation parasite that depends on endogenous bacteria some cats remain infected for life.
5) Trichomonas foetus is a species of single flagellated parasites that is known to be after the abortion of the fetus & the cow's return to a normal estrous cycle, the row may come into estrus.




- Pathogenicity :-

The infection are usually causes during copulation ones bull becomes infected the infection becomes permanent & can be transmitted to other cows during mating like some other vertebrates mammals like cattle, sheep horse, can also become infected with this flagellate parasite. If the cow are infected. It result in abortion by means of distraction of placental attachment & removal of aborted foetus that are retained within the uterus.

- Diagnosis :-

1) Diagnosis may rely on microscopic examination of vaginal or preputial smears.
2) The 5.85 rRNA gene of T. foetus was found to have 12 copies in the T. foetus genome.
3) Complementary fixation can be performed to detect parasite anitibodies in vaginal secretions.
4) Diagnosis can also be done using the in pouch. TF from a prepuce scraping sample from a bull.

- Prophylaxis :-

Again, kept in mind that bulls, remain carriers for life once they become infected, they are unable to clear the infection like cows can. This presents a dilemma since bulls do not show any clinical signs.

The only way to know if a bull is infected is to send a sample to laboratory for testing.

- Treatment :-

Morgan in “1947” reported that the treatment of bulls with sodium Iodide (NaI) more recently for treatment antiaramyein & teramycin are used for treatment. But main infection are best at weakly intervals to overcome trichomonus foetus infection.

# 03 PARASITIC PLATYHELMINTHES (TREMATODES)

Contents

## 1) Introduction :-

- Phylum :- Platyhelminthes.
- Class :- Trematoda.
- flat worm.
- Infection results in a liver fluke.
- Has important economic impacts around the world.
- Rarely infects humans

Trematodes are of extreme economic importance, specialy in tropical developing countries. There is very considerable economic doss in the form of mortality, reduced weight gain decreased production, low fertility reduction in draft power and poor hide quality in Bangladesh due to parasitic diseases.

Different species of vector snails carry larval stage of different trematodes belonging to the superfumilies schistosomatoidae fascioloidea, paramphistomoidea. Etinastomatoidea, Diplostomoidea & pronocephaloids.

They are internal parasite of mulluses and vertebrates. Most trematodes have a complex life cycle with at least two hosts. The primary host where the flukes sexually reproduce is a vertebrate the intermediate host, in which asexual reproduction occurs is usually a snail.

## Systematic Position :-

Kingdom - Animalia
Phylum - Platyhelminthes
Class - Rhabditophora
Super order - Neodermata
Clade - Trematoda

## 2) Classification of trematodes :-

- Blood flukes
  - *Schistosoma mansoni*
  - *S. Japonicum*
  - *S. hematobium*
- Intestinal fluke
  - *Fasciolopsis hepatica*
  - *F. buski*
- Liver fluke
  - *Clonorchis sinensis*
  - *Opistorchis*
- Lung fluke
  - *Paragonimus Westermani*

## 3) General organization of trematodes :-

1) These are leaf-shaped unsegmented flat worms, called flukes.
2) Size varies from 1 mm to several centimeters in length.
3) The organs of attachment are two strong muscular cup-shaped depressions, called 'suckers' the one surrounding the mouth is called the oral sucker & the other, on the ventral surface of the body, is called the ventral sucker.
4) Sexes are not separate i.e. each individual worm is a hermaphrodite (monocious) except the schistosomes which are unisexual.
5) Trematoda is a clade within the phylum platyhelminthes. It includes two groups parasitic flatworms known as flukes. They are internal parasites of mouluscs and vertebrates most trematodes have a complex life cycle with at least two hosts.

6) Class trematoda :- There are about 900 species of trematodes all of which are parasitic most parasitize vertebrates.
7) Adaptation for parasitism include suckers & hooks for attachment glands to produce cyst material & increased reproductive capacity.
8) Class trematoda structurally trematodes are similar to turbellarians having a well developed digestive system and similar nervous, excretory and reproductive systems.
9) It is also the surface of gas exchange : There are no respiratory organs and there are usually two testes, with sperm ducts that join together on the underside of the front half of the animal.
10) The pharynx connects, via short oesophagus to one or two blind-ending caeca, which occupy most of the length of the body.
11) Most trematodes are simultaneous hermaphrodites having both male & female organs.
12) It is connected via, pair of ducts to a number of vitelline glands on either side of the body, that produce yolk cells.
13) After the egg is surrounded by yolk cells, its shell is formed from the secretion of another gland called mehlis gland or shell gland, the duct of which also opens in the Ootype.
14) The Ootype is connected to an elongated uterus that opens to the exterior in the genital pore close to the male opening.
15) Released from the egg is the miracidium. This infects the first intermediate host in one of two ways either active or passive transmission.

16) The sporocyst forms inside the snail first intermediate host & feeds through diffusion across the tegument.
17) The rediae also forms inside the snails first intermediate host & feeds through a developed pharynx.
18) Either the rediae or the sporocyst develops into the carcariae through polyembrony in the snail.
19) The mouth is located at the forward end of the animal & opens into a muscular pumping pharynx.
20) The ovary is sometimes also associated with a storage sac for sperm and a copulatory duct termed Laurer's canal.

## 1) SCHISTOSOMA HAEMATOBIUM

- **Systematic position :-**

Phylum - Platyhelminthes

Class - Trematoda

Order - Diplostomida

Family - Schistosomatidae

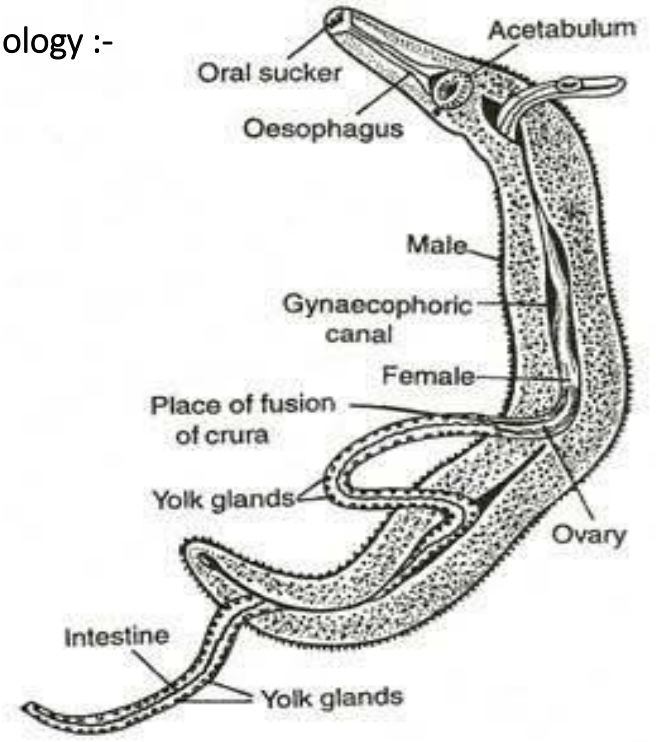
Genus - Schistosoma

Species - S. Haematobium

**Geographical Distribution :-**

Schistosoma haematobium lives in the total vessels of man. It is cosmopolitan in distribution. It is common in Africa any parts and typically America including Westindies, particularly in Egypt, South Western Asia. A few cases from Ratnagiri in Maharashtra State to India.

**Morphology :-**



It is commonly called as blood fluk of man, but in old days it wall called as “Biharzia” in man it causes a disease is known as “Biharziasis” or “schistosomiasis” internation committee of Zoology nomenclature but W.H.O. still calling it “Biharzia”.

It structure the three / 4 species of adult worms resembles each other closely. These are present in the flood vessels, schistosoma having long living life span is 20-30 years.

### Colour :

Body colour of the parasite is grayish or pinkish in colour. But the present in colour due to degenerating blood cells of host.

### Shape & size :

Body of S. haematobium is thin cylindrical finely tuberculated. The male animal shows 15 mm in length & female animal shows 20 mm in length, the male are cylindrical and thick white female are long & thin.

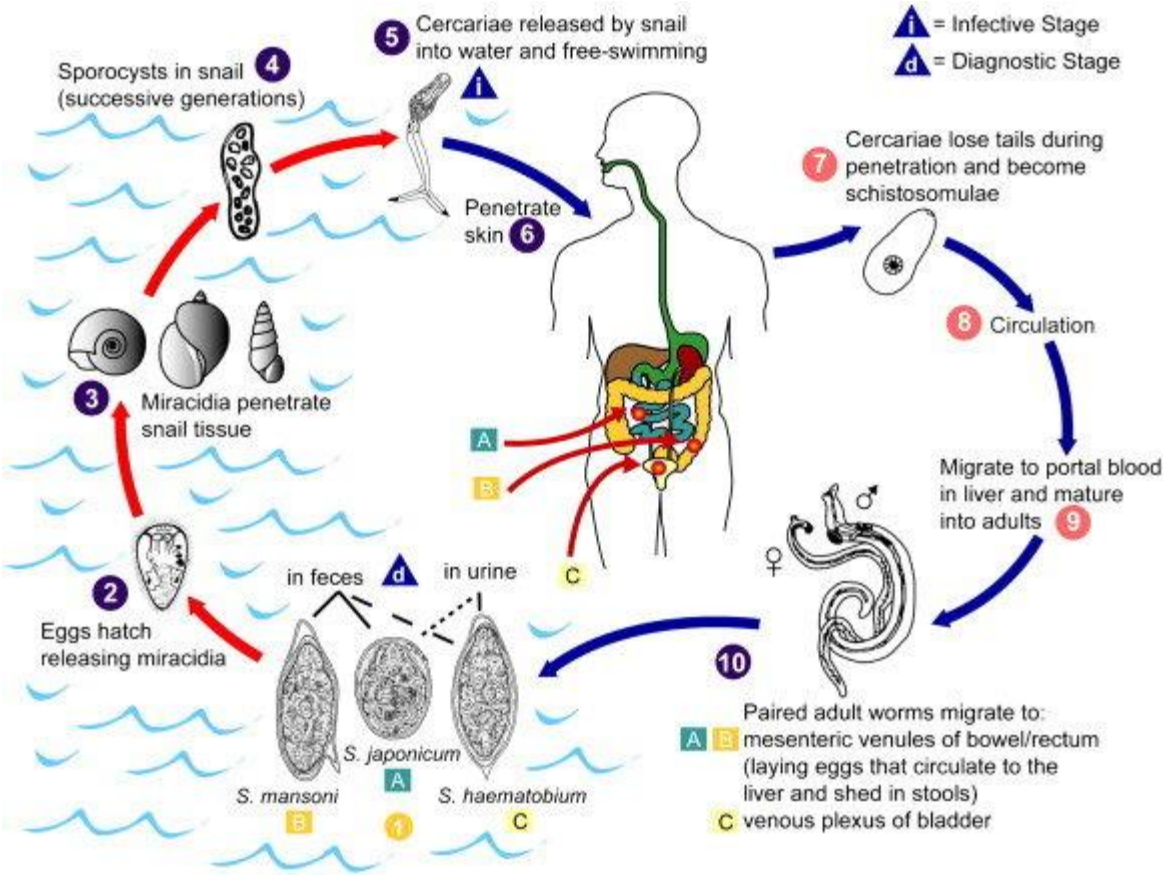
### Suckers :

In both the sexes of the anterior end there is a small oral sucker which surrounds the mouth, aperture. The another larger posterlorly situated sucker on acetabulum is present ventrally close to oral suckers.

### Gynocophoric canal :-

s already stated, schistosoma unisexual, the male is smaller than female which is long and delicate the ventral side of males body is deeply infolded to form the gynocophoric canal in which the female is lodged.

Life-Cycle :-



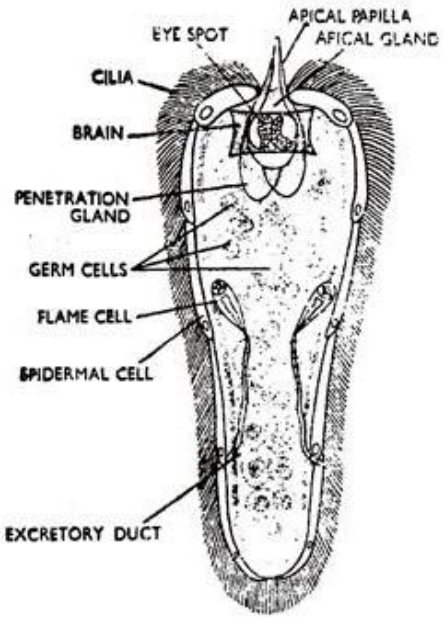

It is a diagenetic parasite which requires two hosts, primary and final host, the final host is man & secondary host is fresh water snail.

Mode of transmission :-

As the male & females are closely associated in the vessels of man, her it takes place copulation, fertilization is intemaly. The fertilization is occur in the oviduct. After fertilization eggs are formed. This eggs undergoes secretion of mehlis, gland after secretion of outer covering is form, it is called egg capsule. This are measuring ½ to 170 micron in length & 40-70 micron in width & this egg is light brown in colour & it shows spines with this

eggs bears a spine which facilitates it penetration into the intestine or urinary bladder, finally they come out with urine body of man.

When the eggs are comes in contact with water they hatch & miracidium larva is liberated.




It measures 130 micron in length and 60 micron in width. The larva contains ciliated epithelial covering. It is free swimming larva, it shows promative gut & two pairs of flame cells, this larva shows many germ cells. It's life is very little it swims very actively in the water in search of secondary host (ile Bulinus).

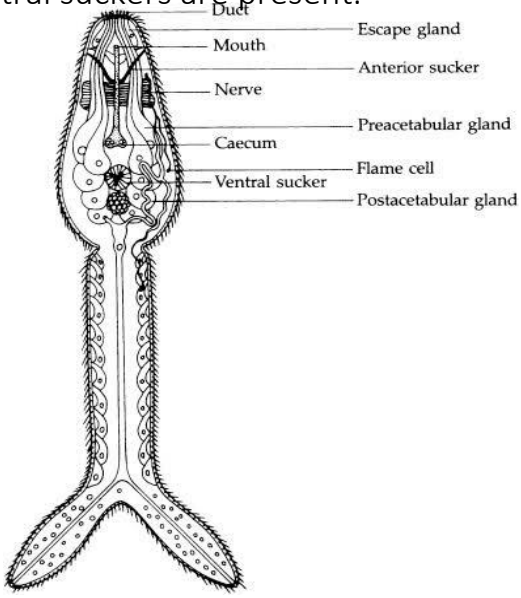
Transmission into secondary host :-

When larva enters into the body of the snail, here it boving the soft tissue of the snail and then enters into the liver here, losses cilia. It is modified into sporocyst the sporocyst is developed second generation sporocyst by pathogenesis the second generation sporocyst produce cercaria by

pathogenesis but in schistosoma haematobium redia larva is absent the sporocyst and escape from the snail into water.

## Cercaria Larva :-

The body of cercaria is short and oval it has long forked to tail both covered with minute, spine oral & ventral suckers are present.




## Transmission into definite host (Man) :-

When the cercaria Larva comes in the contact with skin of human beings in the pond water here, it enters into blood, capillaries know this such cercaria called schistozomulaes and again they enter into perlpheral vennules from here they carried through the right side of the heat into the pulmonary capillary. They remain in the capillaries for some days them they are carry to the left heart & then into the systematic circulation, it requires five days to reach the liver in the infrahepatic portion of the portal blood stream the larva grows into adult all the larva produced from one egg becomes adult of the some sex no female worm is matured in host.

It takes about 1 to 3 month for the worm to reach vesicle & pelvic plexus of veins after the initial exposure of the skin. When the worms are become sexually mature they copulated the females are enclosed in the males & the fertilized egg which are ultimately comes out with urine thus the cycle is gets repeated.

## Pathogenicity :-

When cercaria penetrates the skin it causes irritation and itching called as 'swimmers iteh' so it causes a disease is known as schistosomiasis. The schistosoma eggs are migrating through tissue to rich the column of the urinary bladder tissue reaction is form of inflammation connective tissue in copsulation formation takes place.

The terminal spinal eggs migrating through the wall of urinary bladder sometime it becomes the blood vessels so it causes haemorraghe generally occurs they various infections, called schistosomiasis. Bihariasis, is case of heavy infection, the mature worm migrate to the brain, lungs uterus and gonads.

At lateral stage, blood comes out in urine it is called "Haematuria".

## Diagnosis :-

### Direct parasitological methods :-

1) Detection of S. haematobium eggs in urine.
2) Test for viability.
3) Detection of S. mansoni eggs in stool by direct smear method or by concentration method.
4) Kato thick faecal smear, for egg counting to assess the intensity of infection.

5) Rectal swab.

## Blood examination :-

1) Eosinophilia, leucocytosis, anaemia.

## Prophlaxis :-

1) Eradication of intermediate molluscan hosts.
2) Prevention or environmental pollution with urine & faeces.
3) Effective treated of infected.
4) Avoid swimming, bathing & washing in infected water.

## Treatment :-

The drugs having specific actions on the schistosomes are nitrothiazole compound niridazole, nilodin, hycanthone, trivalent antimony compounds, such as tartar emetic, fouadin, anthiomaline, antimony dimercapto-succinate.

## 2) PARAGONIMUS WESTERMANI :-

### Systematic position :-

Phylum - Platyhelminthes

Class - Rhabditophora

Order - Plagiorchiida

Sub-Family- Traglotrematoda

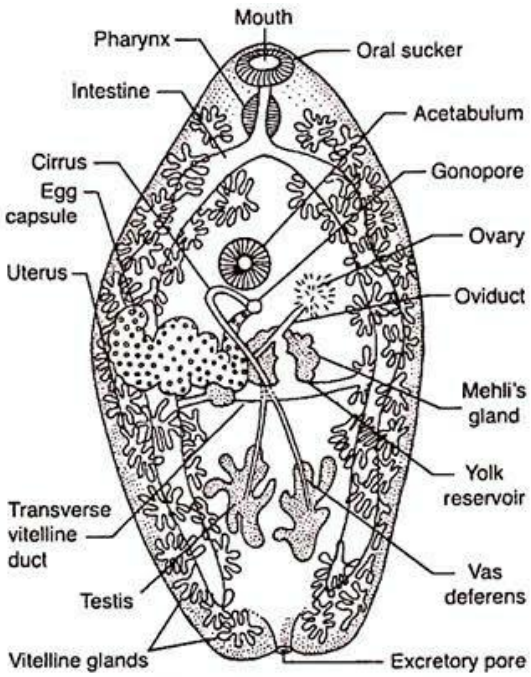
Genus - Paragonimus

Species - P. Westermani

### Geographical Distribution :-

The organism is dristributed in china, Japan, Jaiwan, Korea, the soviet union, phillipines, Thailand, Indonesia, India and Shrilanka. In Korea alone 1.0 and 1.5 between million people are infected.

- ### Morphology :

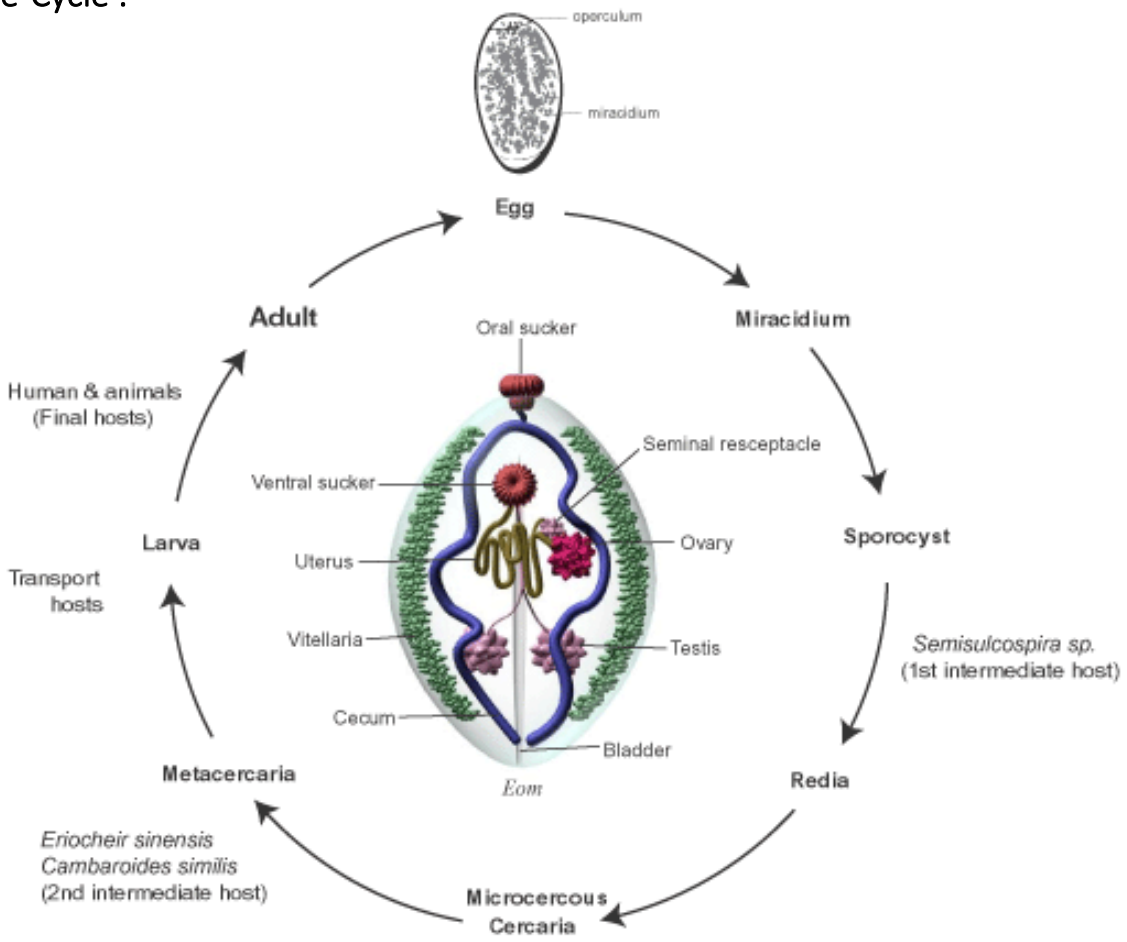


**Adult Warm :**It is thick fleshy and egg. Shaped. Its anterior end is slightly broader than the posterior end. It measure 8 to 12 mm in length by 4 to 6 mm in breadth & 3 to 5 mm in thickness. The ventral sucker is situated near about the middle of the body. The excretory vesicle is large and extends from the posterior extremity to the anterior region, dividing the body into equal halves. The two blind intestinal caeca are unbranched and extend to the caudal region. The genital apparatus follows the same general pattern of trematodes.

**Life Span :-** of the adult worm is about 6 to 7 years.

**Eggs :-** These are golden brown in colour, oval in shape & provided with flattened opercula. They measure 80 μm by 55 μm and each egg contains an unsegmented ovum surrounded by yolk cells.

**Life-Cycle :-**



P. Westermani passes its life-cycle in three hosts –

(1) Definitive &

(2) Two Intermediate host

| First | Second |
|---|---|
| Host | Host |

**Definitive Hosts :-** Man and domestic animals usual hosts in Asia are the tiger and leopard feline hosts serves as reservoirs of infections.

**Intermediate Hosts :-**

**First host :-** A fresh-water snail of the genus melania.

**Second host :-** A fresh-water crayfish or a crab.

The adult worms live in the respiratory tract of definitive hosts. The eggs generally escape in the sputum & some are eliminated in the faeces. In water, a ciliated embryo (miracidium) develops inside the egg in 2 to 7 weeks time on attaining maturity, the miracidium escape into water and swims about in search of its snail host, a species of the genus melania. Inside the soft tissue of the snail the miracidium casts off its tail and passes through the stages of sporocyst & two generation of radiae being finally transformed into cercariae, the whole cycle taking about 10 to 12 weeks. The mature cercariae escape from the snail into water & enter into its second intermediate host, a frest-water crab or a crayfish. Inside the crustacean host they become encysted in the viscera, muscles & gills.

When the raw flesh of an infected crab or crayfish is eaten by man and other susceptible hosts, the cyst-wall is dissolved by the gastric juice and the adolescaria is released in the duodenum. These young worms penetrate the wall of the small intestine & enter the abdominal cavity. Later, they migrate upwards, piercing through the diapharagm & the two layers of the pleura, to gain entrance into the lungs where they finally settle & grow to sexual maturity (taking a period of two weeks for such migration). Eggs are discharged into a bronchiole and are coughed out with the sputum. The cycle is repeated.

Pathogenicity :-

Infection with P. Westermani is known as paragonimiasis.

Mode of infection – Eating of raw or improperly cooked flesh of an infected crab or crayfish strips of raw crab meat soaked in alcohol k.a. “drunken crab” is a popular delicacy in china.

Infecting Agent :- Metacercaria or adoles caria inside a cyst.

Portal entry :- Digestive tract.

Side of localization :- Lungs.

The adult worm as it moves around causes lesion (worm cysts & burrows) by mechanical damage. The eggs also excite a foreign body granulomatus reaction which may soften to fibrous granulation tissue (epithelica cells, lymphocytes, plasma cells, eosinophils, giant cells and fibroblasts).

Prophylaxis :- (control measure)

The measure include –

i) Disinfection of the sputum & the faeces.

ii) Eradication of molluscan host.

iii) A voidance of the consumption of raw freshly salted or inadequately cooked crab & crayfish as food.

Treatment :

Emetine and chloroquine have both been used for paragonimiasis but neither is a good remedy. Bithinol, a dischlorophene derivative has been found by yokogawa and De Jongh (1961) to give encouraging result.

## 3) GASTRODISCOIDES HOMINIS :-

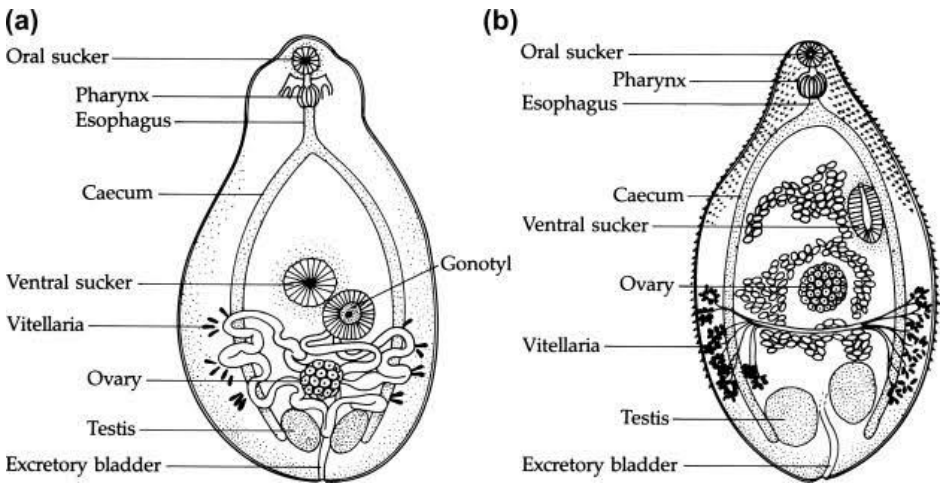
### Systematic position :-

Phylum - Platyhelminthes
Class - Trematoda
Order - Plagiorchiida
Family - Paramphistomidae
Genus - Gastrodiscoides
Species - G. hominis

### Geographical Distribution :-

G. hominis is most common in India & Assam but also occurs in cohin, china and malya state. It has been meet with in India emigrants to british guina.

### Morphology :-



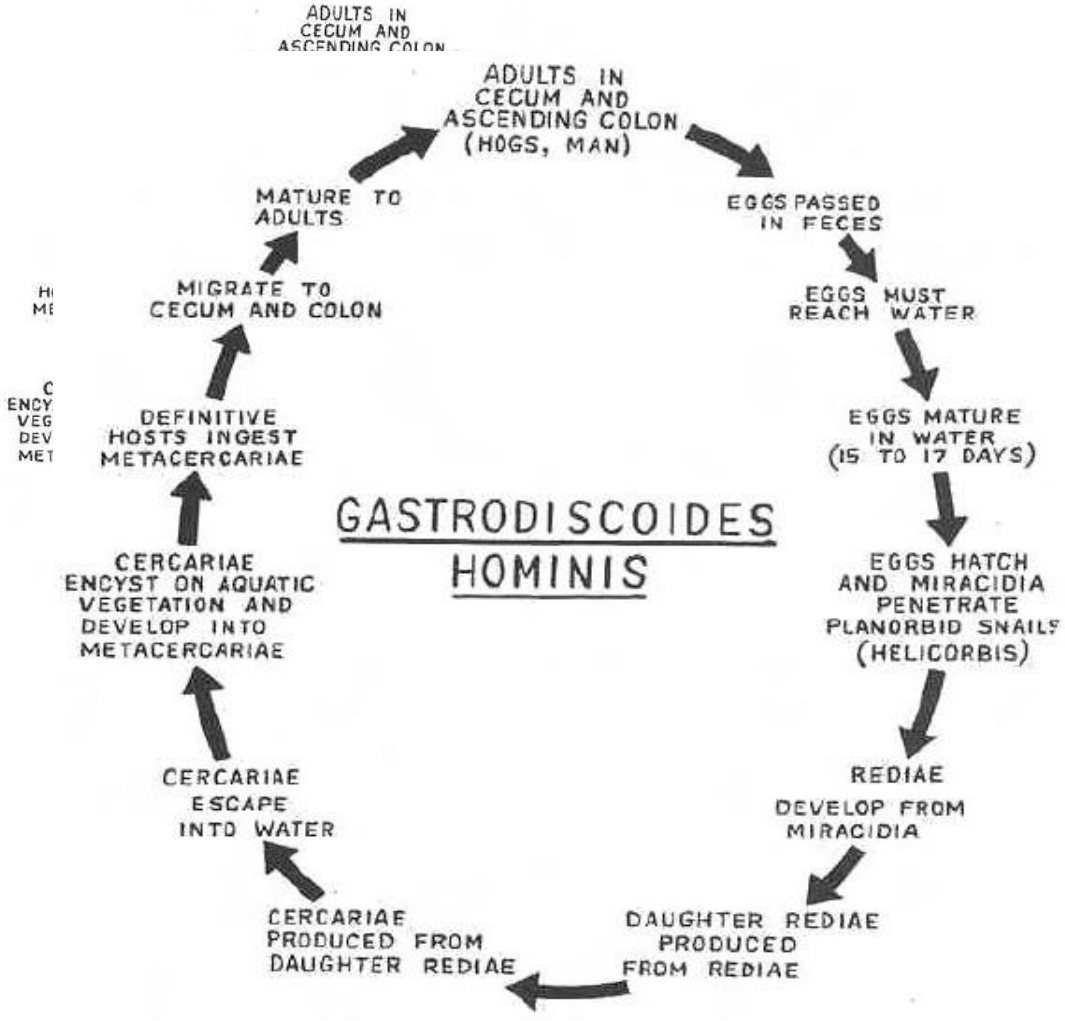

The adult worms are reddish brown puriform flukes with a convex dorsal and concave ventral surface.

It measures 5 to 6 mm in length and 10 mm in breadth. There is a very small oral sucker at the anterior extremity of the body & a very large. Ventral sucker at the posterior extremity. The surface of the body is devoids of spines. The body is demarked into a narrow cone anteriorly. The pharynx is

provided with a pair of lateral pouches and a posterior bulb. The intestinal caeca extend to the middle of the disc's A elongate excretory bladder lies above the ventral suckers and leads to the posterior extremity pore. Genitelia are confined to the discs. The testis are large paired lobate and situated at the anterior side of the disc.

Just behind the bifurcation's of the intestinal caeca. There is no cirrus the single oval overy is smaller than the testis and is situated behind them. The viteline gland's are of fan shaped groups near the lateral margin's of the disc the uterus & the seminal duct open on a prominent anterior genital cone laurer's canal is present.

- Life Cycle :-



The normal hosts of this fluk are apparently man, the pig & the mouse, deer, the adult fluke accurs attached to the wall of the caecum & scending colon.

This sticky greenish brown egg's which are ovoid, operculate and measures 150 by 60 to 70 microns pass out in the faeces of the host, the requires a period of embryonations and develops to hatching stage in to two and a half weeks. At the optimum temperature of 27°C to 34°C. The miracidium lack's eye spots but is never the less phototactic.

It is a long narrow organism's with a prominent apical papilla. The rest of the life history is unknown. But the releated species gastrodiscus aegytiacus a parasite of Eguidae utilize snail of the genus cleoptera or intermediate host and emerging cercariae encyst's on vegetation & a water the snail could not well be a species of coleopteran as this genus is sticky African but it might belongs to the same family thioridae. It has been suggested that the cercariae of gastodiscoides hominis may encysts in amphibians and other lower vertebrates other than a vegetation.

## Pathogenicity :-

G. hominis, produces some irritation of the ceacum & colon with resulting mucous diarrhea paragnosis is good immunity (not studied).

## Diagnosis :-

The finding of the characterstics egg in the faeces is conclusive.

## Prophylaxis :-

Before precise prevention measures can be devised the life-cycle must be educidated and the epidomiblogy unfiltered or unboiled water and from eating raw vegetables and other foods probably constitute satisfactory protective measured for the individuals.

## Treatment :-

Thymole and high soap saline enameta have been successfully used Hexyl, resorcinol or tetrachloroethylene would probably also be effective.

## 4) FASCIOLA HEPATICA :-

### Systematic position :-

Phylum - Platyhelminthes

Class - Rhabditophora

Order - Plagiorchiida

Family - Fasciolidae

Genus - Fasciola

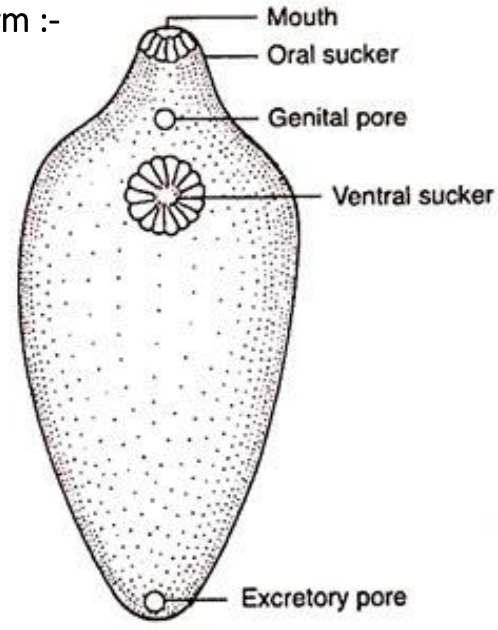
Species - F. hepatica

### Geographical Distribution :-

The common names of F. hepatica are 'the sheep liver fluke' and 'the common liveefluke'. It was the first trematode to be described. It has a consmpolitan distribution and is prevalent in most sheep-raising countries. Adult worms, live in the biliary passages of the liver of sheep cattle, coat & man.

### Morphology :-

i) Adult Warm :-



It is a large leaf-shaped fluke, measuring 30 mm long & 15 mm broad and is brown or grey in colour. It is bilaterally symmetrical and has no true body cavity. It has a conical projection at anterior end & is rounded posteriorly.

The oral sucker is situated in the conical projection at the anterior end whereas the ventral sucker is present nearby in line with two shoulders. The intestional caeca and testes are extensively branched. Life span of adult parasite is about 10 years.

ii) Eggs :-

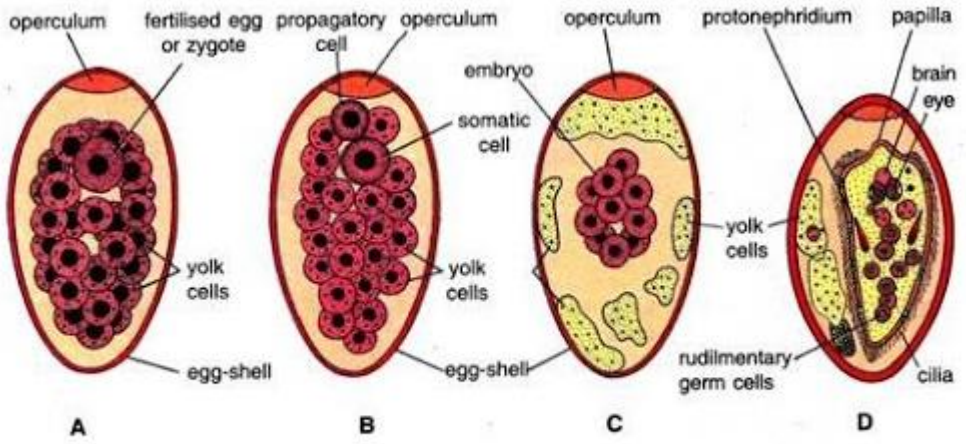
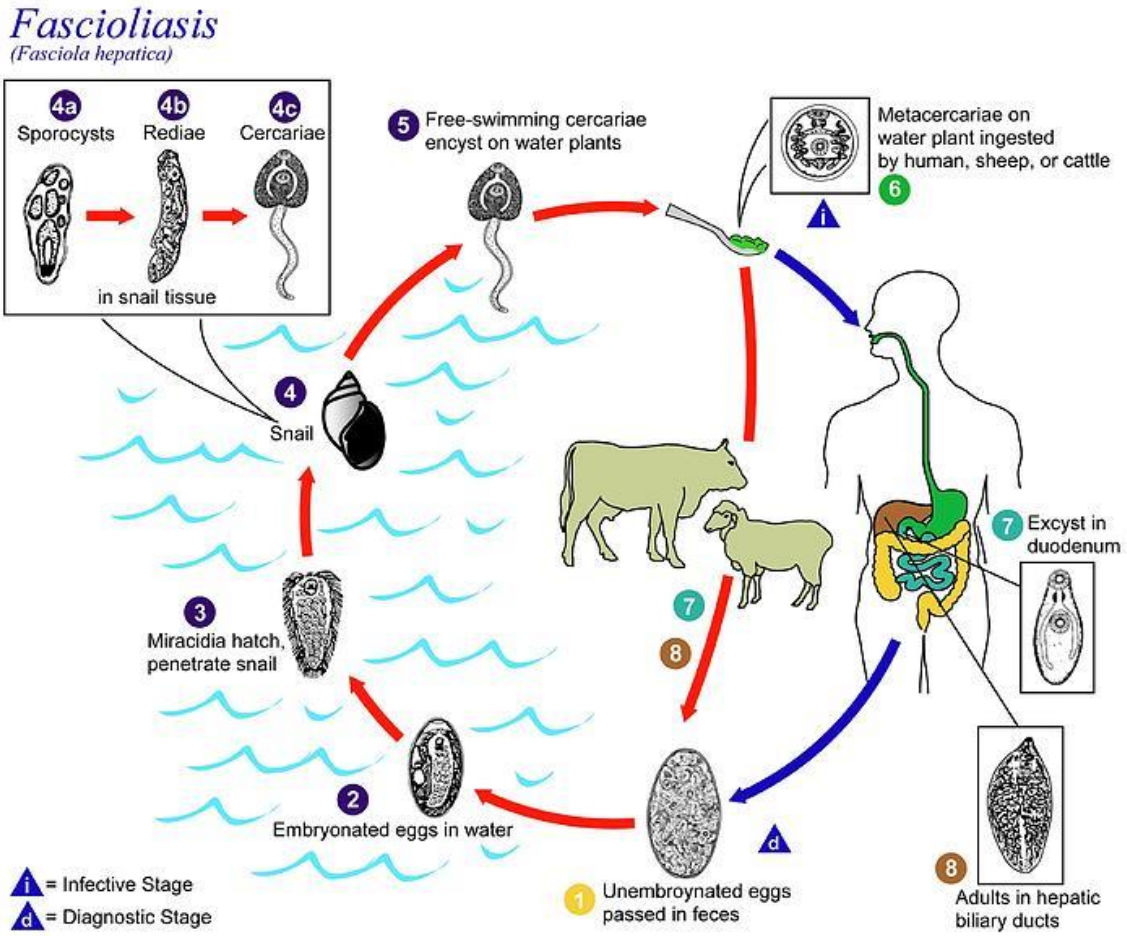



**Fig.** *Fasciola hepatica.* Early stages of development. A—Fertilised egg; B—Two cell stage; C—Many cell stage; D—Miracidium in capsules.

The egg is large, oval, operculated, bile stained & measures about 140 μm x 80 μm. Each egg contains an immature larva, the miracidium. The shell of egg is thin with a smooth surface. Egg cannot be differenticated from those fasciolopsis buski.

Life-Cycle :-




F. hepatica passes its life-cycle in one definitive and two intermediate hosts.

**Definitive hosts :-** Sheep, cattle, goat & man.

**First intermediate host :-** Snails of the genus lymnaea.

**Second intermediate host :-** Aquatic vegetation e.g. watercress.

**Infective form :-** Metacercariae encysted on water plants.

The adult worms live worms live in the biliary passages of the liver of the definitive host. Eggs are passed out in the faeces. These eggs enters

the freshwater (Lakes, canals etc.). The embryo matures in water in about 10 days and a free-swimming miracidium is released. It penetrates the tissue of first intermediate hosts. Snails of the genus Lymnaea. The miracidium she its ciliated cover & passess through the stages of sporocyst, the first and second generation redia stages to become the straight tailed cercaria in about one to two months. The cercaria escaeps from the snail into water and encyst on aquatic vegetation to become infective metacercaria. Sheep, cattle, goat or human acquire infection by ingestion of uncooked aquatic plants containing the metacercariae.

The metacercariae exayst in the duodenum and pierce the gut wall to enter the peritoneal cavity. They penetrate the capsule of the liver, traverse the liver parenchyma & ultimately settle in the biliary passages, where they mature into the adult worms in about 3-4 months. The adult worm beings producing eggs which exsist in the faeces. The life cycle is thus repeated.

Pathogenicity :-

Fascioliasis (F. hepatica infection) occurs mainly in rural area. It is most common among sheep & cattle herders. Injury to liver parenchyma occurs by the young migrating flukes. This is accompanied by fever, abdominal pain, tender hepatomegaly, ascites & jaundice. Ascitic fluid contains eosinophils & other leucocytes.

Larvae migrating through the peritoneal cavity may become lodged in ectopic foci & lead to formation of abscesses. These sites include flood vessels, lungs, subcutaneous tissue, brain & the orbit.

- Diagnosis :-

i) Demonstration of eggs in stool or in bile.

ii) Moderate to high eosinophilia is constantly present.

iii) Serological tests have been used for detection of antibody. ELISA is a sensitive method & becomes positive within two weeks of infections. It become negative after treatment.

- Prophylaxis :-

Preventive measure include –

i) Health education.

ii) Avoidance of eating raw or uncooked aquatic vegetations.

iii) Treatment of infected animals.

iv) Destruction of molluscan hosts by use of molluscicides.

- Treatment :-

Oral bithional is the treatment of choice. Thialabendazole & dehydroemetine are other drugs used for the treatment.

### III) Parasitic Adaptation in Trematodes :-

To adapt to this microenvironment certain morphological, anatomical & physiological changes occur and because of which the parasite survives in the host. Such cnages which facilitate a parasite to adapt to parasitic mode of life in the host itself are teguent in taenia called parasitic adaptation.

Adaptation is fitness of an organism to its environment the parasitic nematodes cestodes are trematodes particularly have undergone profound adaptation to suit their parasite mode of life as it is stated that their adaptation are morphologically as well as physiologically forms.

### I) Morphological Adaptation :-

### i) Body covering :-

The body of parasite is covered by a thick covering called “tegument”. A tegument is also with scales to give protection to the parasite. The tegument is probably a protoplasmic layer but continuously and renewed by cells.

### ii) The Shape of body :-

Generally the shape of body is small, flat, dorso ventrally flattened, leaf like or ribbon like as it is found in most of trematodes.

### iii) Organs of body :-

For the grip and the attachment to the body of host & tissue parasites have some special organs of attachment.

The flatworm like trematodes for this purpose are various arms with suckers hooks and spines. Then sucker may be with or without hooks and spines.

### iv) Organs of Locomotion :-

Parasites are without organs of locomotion because parasites leave in the body of host where sufficient food is available without any effort. Thus the organs of Locomotion such as cilia, flagella, etc. are absent. Without any effort and movement they can even reproduce, because other opposite sex individuals are available.

### v) Organs of Nutrition :-

Being parasite mode of nutrition the food of the parasite is already digested food and semidigested forms. Elaborate organs of nutrition is does not needed. Trematode have and incomplete growth with suctorial pharynx & cephalic suckers for sucking the blood.

### vi) Neuro sensory system :-

It is required by the free living animals for quick and efficient response to stimulate. But it is not required for the parasites parasitic life is in a scale

environment. Hence there is a profound reduction of nervous system and the total absence of sense organs. In nematodes it is some what developed because of it's complicated life history and hosts.

vii) Reproductive system :-

Reproductive system is enormously developed in the parasite it may help to produce the large number of eggs, inspites of various complication of life histories. There are better chances of survival and maintain continuity of race.

Generally the basic plan of reproduction system is monoecious or hermaphrodite, hermaphroditism is of distinct advantage to the parasite. Because self copulation is possible and after copulation both the individuals by lay egg and thus doubling the rate of reproduction.

II) Physiological Adaptation :-

i) Protective mechanism :-

The alimentary canal of parasite and the body are variously modified to protect themselves from the action of digestive juices. In tape worms gut wall secretes which forms protective covering around the parasites, trematodes and nematodes also secretes antienzyme to neutralize the digestive enzyme of the host. The other covering of the parasite is called 'cuticle' or 'intugument'. It is tough hard and protective.

ii) Anaerobic respiration :-

Parasite respires anaerobically by breaking down glycogen because environment in the gut & various parts of the body of hosts is without free oxygen.

### iii) Osmoregulation :-

The osmotic pressure of the endoparasites body fluid is almost the same as that of the host. This helps osmoregulation unnecessary except in trematodes.

### iv) High fertility :-

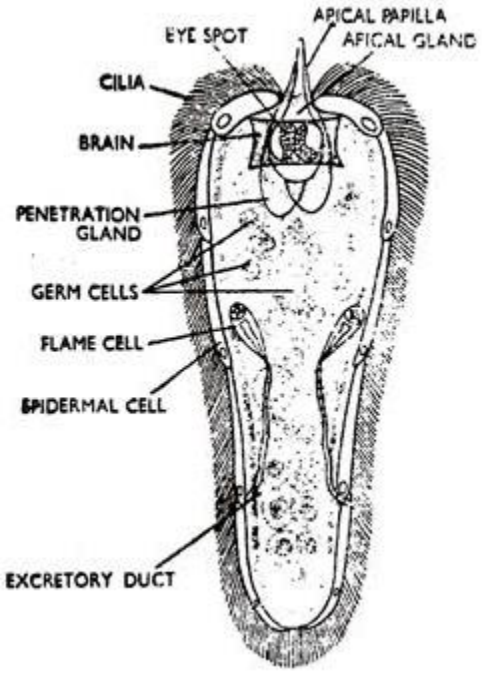
Fertilized egg produced by parasites have very uncertain features it is due to complex life histories hence the parasite are producing large number of ova to bring about fertilization and large number of fertilized eggs are produce through some of the fertilized eggs are destroyed the remaining may develop into adulthood. Thus existence of the species is maintained.

### v) Polyembryony or pedogenesis :-

Polyembryony is the process in which asexual multiplication of larval forms occur. In case of various parasitic trematodes the larva are multiplied asexually such as radia gives rise to larges was of daughter radio from the large germ balls of the parent radia. In the same way a single hexacantha of echinococcus produces several scolices each of which is a potential type worm.

IV) Larval forms in Trematodes :-

1) Miracidium Larva :-




1) Miracidium the first larval stage of trematodes worms (flukes) which hatches from eggs excreted in the faeces of the primary host.
2) It's leaf like body is covered with cilia, enabling the larva to swim towards a secondary host, in which it continues its development.
3) The enzyme dissolves the cementing material by which the operculum is attached, thus the releasing operculum.
4) It is first larval stage in the life cycle of fasciola hepatica.

5) Expansion of the granular cushion, accompanied by exosmosis of salts & other materials from within the eggs pushes off the operculum.

6) Miracidium of fasciola is about 0.07 mm long, oval or conical. It broad anterior end is produced into an apical lobe or apical papilla or terebratorium.

7) Pouch – like multinucleate apical gland.

8) A number of unicellular penetration glands.

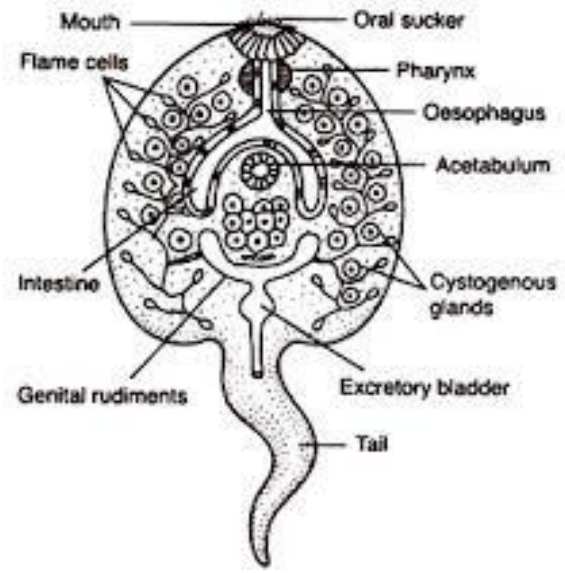
9) An outer layer of circular & inner layer of longitudinal muscles.

10) In addition there is pair of large pigmented, eyespots, a large larval brain & simple nervous system in the anterior part of body.

11) Except the apical lobe, the rest of the body is ciliated.

12) It is covered with 21 closely fitted hexagonal epidermal plates, arranged in five rows.

2) Cercaria Larva :-



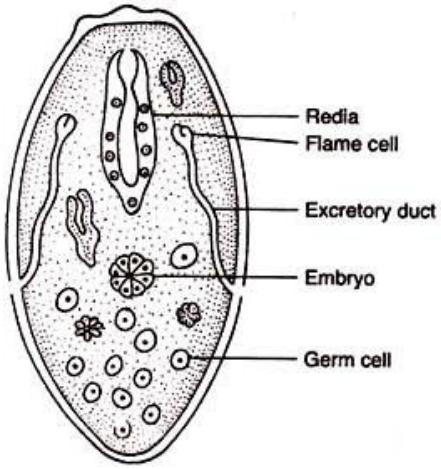

Body of the cercaria is short and oval with a long forked tail, both covered with minute spines oral and ventral suckers are present. The mouth leads into the oesophagus which leads into a trifid intestine, pharynx is absent.

The larva contains an excretory system & nerve elements. There are five pairs of penetration gonads two parts of anterior oxyphilic glands & three pairs of posterior, basophilic glands.

The cercaria larva leave the snail and swim freely in water for some time without encystment mode of infection is by skin penetration or by drinking infected water so that a metacercarial stage is simply omitted. The cercaria larvae may penetrate the skin of a person when wading bathing or washing in the contaminated water. The bore through the human skin quickly to enter the blood capillaries and then reach the liver.

Cercaria larva reach the portal system of the host through lymph vessels, venous blood vessels. Mesenteries and capillaries they feed on blood pair & develop to adult stage. All the larva produced from one egg become adult of the same sex. No female worms mature in a host, if the male worm are absent.

3) Sporocyst Larva :-




It looks like an elongated sac, about 0.7 mm long. Its body wall retains all the layers of body wall of miracidium except the ciliated ectoderm. During metamorphosis into sporocyst, the outer coat of ciliated cells is thrown off the pigment cells of the eye-spot separate from one another & loose their crescentic form & the organism becomes a mere sac.

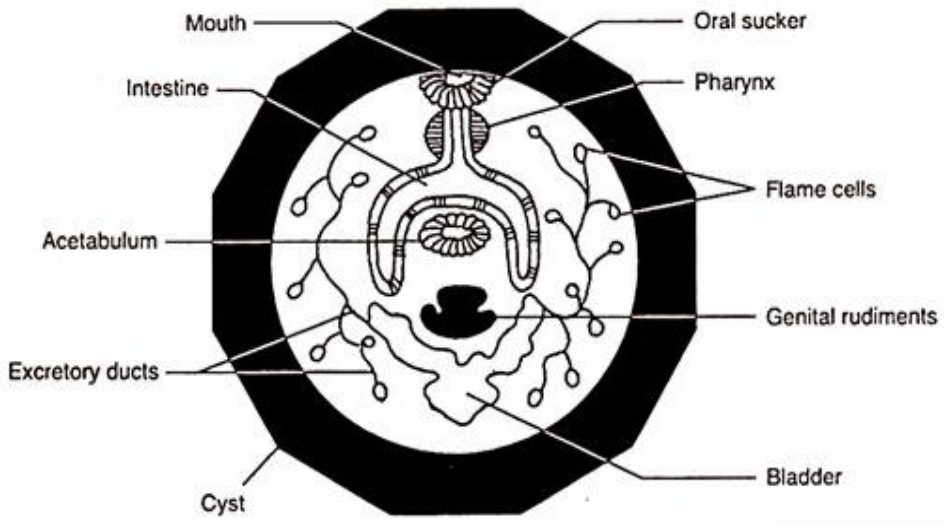
It is known that glands, brain eye spots & apical papilla of miracidium degenerate and disappear in the sporocyst. This sac like stru. Is covered by cuticle.

Protonephridium on each side divides into two flame cells which open outside by a common excretory pare.

1) The sporocyst contains germ cells which by division form Redia Larvae or daughter sporocysts.
2) The Redia larvae when fully formed come out by rupturing the sporocyst wall.

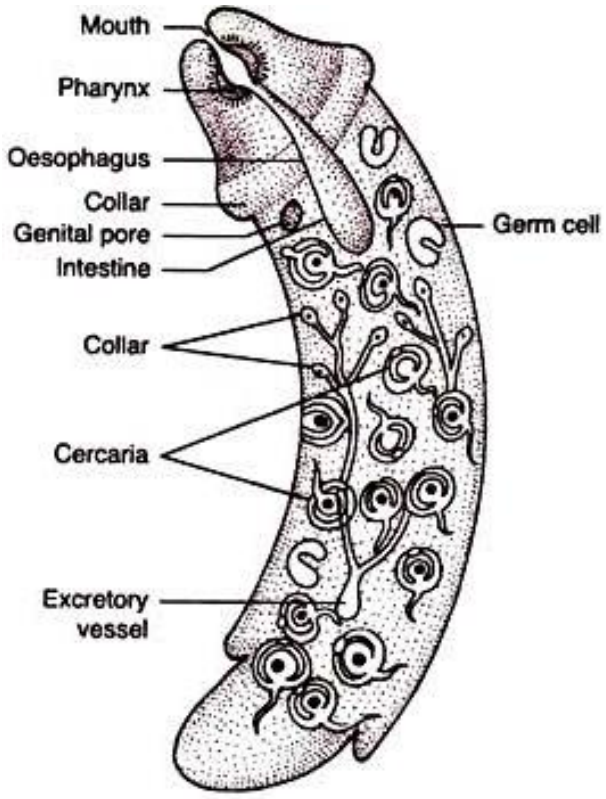
## 4) Metacercaria Larva :

1) When full formed the cercaria attains & length of about 0.28 mm. then it emerges through the birth pore of Redia. With the help of its suckers & contractile tail, it wriggles out of the tissue of the host.
2) It leads a free existence four about 2 or 3 days & it finally gets attached to some aquatic plant & wet grass.




3) Shortly, it ceases to move about and rests on a leaf of the aquatic plant or a blade of the grass.
4) It loses the tail & starts encystment. During encystment the cystogenous cells secrete an abundant slimy material which hardens immediately & in a few minutes the animal is covered by a thick hard cyst & gets firmly attached to the aquatic plants.
5) This encysted larva is called metacercaria.
6) It is in fact the juvenile fluke, also known as marita.
7) Metacercaria differs from cercaria in that if has a rounded form a thick hard cyst wall & large number of flame cells.
8) It lacks a tail & its excretory bladder opens out directly through a single pore. Like cercaria, pharynx & bifid intestine are present.

5) Redia Larva :-




1) Rediae emerge from the sporocyst by rupture of its body wall. Each Redia is an elongated oval hollow body covered with thin cuticle.
2) At the anterior end there is muscular collar.
3) The mouth is at the anterior end which opens into muscular pharynx, which in its turn opens into a small intestine.
4) Little behind the muscular collar is the birth pore.
5) Posterior part of the body has a pair of blunt conical processes called lappets about each lappet lies the excretory pore.
6) The Redia Larva comprises of germ cells which by division produces cercaria larvae in autumn. These come out through birth pore.
7) The rate & extent of development depend mainly on two factors.
8) The available food reserves in the gland.
9) The size of the infection.

# 04 PARASITIC PLATYHELMINTHES (CESTODES)

Contents

## 4.1 Introduction :-

Platyhelminthes (platy means flat; heminth = worm)

1) Tapeworms consist of two main parts; a rounded head celled scolex & a flat body consisting of multiple segments. Each segment is called a proglottid.
2) The scolex has specialized means of attaching to the intestinal wall, namely, suckers, hook or sucking grooves. The worm grows by adding new proglottids from its germinal center next to the scolex.
3) The oldest proglottids at the distal end are gravid & produce many eggs, which are excreted in the feces & transmitted to various intermediate hosts such as cattle, pigs & fish.
4) There are four medically imp. Cestodes – Taenia solium, Taenia saginata, Diphyllobothrium latum & Echninococcus granulosus.
5) Humans usually acquire the infection when undercooked meat or fish containing the larvae is ingested.
6) Tapworms or cestodes are an ancient class of highly specialized flatworm parasites, Adult tapworms inhabit the gut of a vertebrate animal with several species adapted specifically to humans.

## II) Classification :-

### Infecting man

### i) Pseudophyllidean cestodes :-

* Possess false or slit-like grooves called bothria.
* Adult worms in intestine.
    1) Diphyllobothrium latum : fish tapeworm

**Larval stage :** Plerocercoid in man.

1) Sparganum mansoni.
2) Sparganum proliferum

ii) Cyclophyllidean Cestodes :-

* Possess cup life & round suckers called acetabula.

* Adult worms in the Intestine.

Taenia saginata, Taenia solium, Hymenolepis diminuta, Dipylidium caninum.

III) General organization of Cestodes :-

- Cestodes Morphology :-

1) Adult worm white in colour.

2) Very elongated.

3) Body or strobila – flattened.

4) Cephalic portion of strobila differentiated into an attachment organ scolex.

- General Characterstics :-

1) Long, segmented & tape like are called tapeworms.

2) Dorso ventrally flattened.

3) Size varies from a few mm to several meters.

4) Adult worms are found in the human intestines.

5) There are 3 regions in on adult worm :-

(1) Head : scolex. (2) Neck. (3) Strobila (body or trunk).

Consist of series of segment called proglottids.

6) Tapeworms anchor themselves to the inside of the intestine of their host using their scolex, which typically has looks, suckers or both. They have no mouth, but absorb nutrients directly from the hosts gut.

Reproduction :-

1) The neck continually produces proglattids, each one containing a reproductive tracts; mature proglatids are full of eggs and fall off to leave the host, either passively in the faces or actively moving.
2) All tapeworms are hermaphoradites, with each individual having both male and female reproductive organs.
3) The reproductive system includes one or more testes, cirri-vas deferens & seminal vesicles as male organs & a single lobed or unlobed overy with the connecting oviduct & uterus as female organs.
4) The common external opening for both male & female reproductive system is known as genital pore, which is situated at the surface opening of the cup-shaped atrium.
5) Though they are sexually hermaphroditic & cross-fertilization is the norm, self-fertilization sometimes occurs & makes possible the reproduction of worm when it is the only individual in its host's gut.
6) During copulation, the cirri of one individual connect with those of the other through the genital pore & then spermatozoa are exchanged.

- Life-Cycle :-

1) Cestodes are parasites of vertebrates, with each species infecting a single definitive host or group of closely related host species.
2) The definitive host is always a vertebrate but in nearly all cases, one or more intermediate hosts are involved in the life-cycle typically arthropods or other vertebrates.
3) Infection can be long lasting; in humans, tapeworm infection may last as much as 30 years.
4) No asexual phases occur in the life-cycle as they do other flatworms, but the life-cycle pattern has been a crucial criterion for assessing evolution among platyhelminthes.
5) Cestodes produce large number of eggs but each one has a low probability of finding a host.
6) In the pseudophyllidea, many eggs are released in the brief period when their aquatic intermediate hosts are abundant (semelparity).

7) In contrast in the terrestrial cyclophyllidea, proglottids are released steadily over a period of years, or as long as their host lives (interoparity).
8) If the eggs are laid in water, they develop into free-swimming oncosphere larvae.
9) The intermediate hosts are copepods & small fish & the definitive hosts are water birds.

## 1) TAENIA SAGINATA :-

- **Systematic position :-**

Phylum - Platyhelminthes

Class - Cestoda

Sub-class - Eucestoda

Order - Cyclophyllidea

Family - Taeniidae

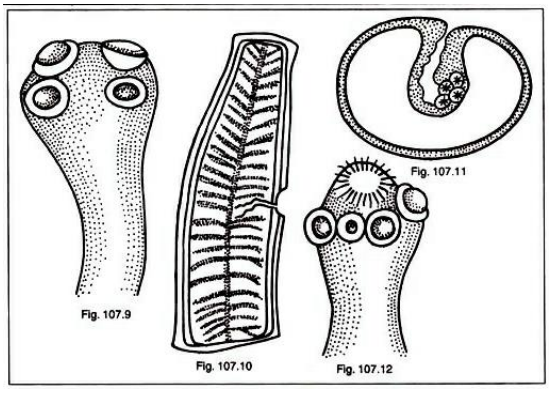
Genus - Taenia

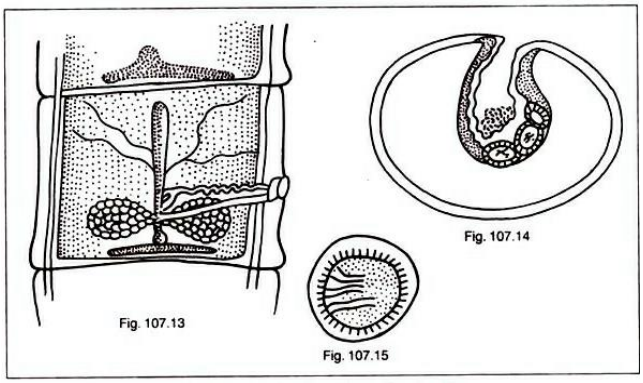
Species - T. Saginata

### Geographical Distribution :-

It is commonly called as beef type worm it is cosmopolital in distribution whenever beef is eaten in India it is more common Mommadians or among hindus community who eats beef.

### Morphology :



Taenia saginata body is elongated ribben like dorsoventrally flattend much larger than Taenia solium it is whitish in colour and semi transparent and measuring 5 to 10 meter in length in some cases it may up to 25 meters are more in length its body divided into three main parts scolex, neck, trobila or body parts.

### Scolex :-

The most anterior tip of the body is called as head it is also called as scolex it is measuring 1 to 2 mm in diameter. It looks like pin head like structure it has four circular suckers the head is without rostellum with hooks hence, it is called unarmed tapeworm below the scolex this is long and narrow part called neck.

### Neck :-

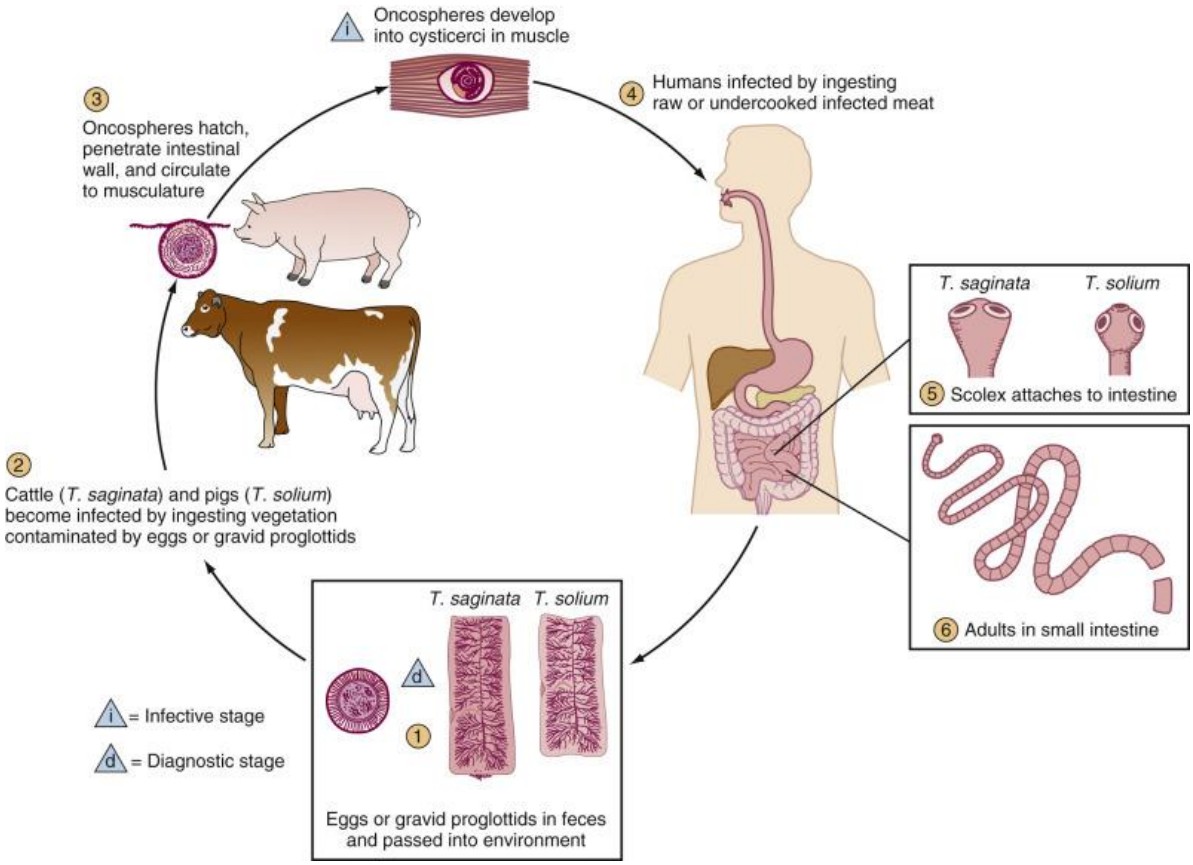
Neck is a long narrow part like duct is it produce posteriorly number of segments called as strobilization.

### Strobila :-

The third part is called as proglottid gravid it is also called strobila the strobila shows 1000 to 2000 segments or proglottids. Proglottids are of three types. The upper two segments are called immature proglottid. The second type of proglottid is called mature proglottid. This show well developed reproductive organs. They show male and female organs while the posterior gravid proglottid having stem like uterus with 15 to 30 in which the fertilized eggs are stored.

The third part is gravid proglottids are cut off or detached from the mother body & pass into intestine of host ile man, the proglattids are cut off by the process called Apolysis.

## Life-Cycle :




It is a digenetic parasite it requires two host to complete is life cycle the first definite host is man and secondary or intermediate host is cattle ile cows, buffallow, bulls etc. bolb host are worm blooded, vertebrates.

## Mode of Transmission :-

When the adult worm lives in the small intestine of man here it undergoes copulation is brought about by folding strobila clefts mating or copulation exchange of gamets and fertilization take place, in the oviduct. The fertilized egg are passed in to secretion of viteline gland after secretion these eggs passes into the uterus of gravid proglottid. These proglottid are cut of from

parasite body and reaches into the intestine of host. Finally the host send outside by the body along with foecal matter.

Transmittion into secondary host :- (i.e. cattle)

When the cattles are sollowing gravid proglottid of fertilized eggs with fecal matter. It reaches to alimentary canal and cattle, here the egg shell is punctured and the larva is liberated it is called “Anchosphere”. The anchosphere stage shows six hooked embryo called hexacanth embryo. It is covered by four covering. These anchosphere penetrates into the gut wall of host with hooks. Now, this larva passes into portal vessel, masentric, lymphatic and finally reaches to systematic circulation usually these embryo travel vim portal vein successfully reach to following organs, like liver, right side of heart, lungs, left side of heart & systematic circulation.

During the course of migration the embryo loses the outer covering and anchosphere are comes in contact with muscular tissue. They ultimetly settle down the undergoes further development generally the anchosphere select tongue, neck, shoulder, for settlement for further development, the anchosphere now loss there hooks and within eight days they gives rise to another bladder like larva forms, it is called cesticercus larva it has a bladder with one scolex the scolex remain within the cyst wall it takes nearly 60 to 70 days per modified the anchosphere stages to cesticercus stages.

The cysticercus is about 5 to 10 mm in length & 3 to 4 mm width it contains anamed scolex evaginated at one side it can live for about eight month in flash in cattles ile beef.

## Transmission into definite host (Man) :

It can only develop further when ingested by man the cysticercus larval stages is important in the life cycle in Taenia saginata it is called “cysticercus boris” it is an infective form. The infection causes to man when he eats under cooked beef containing cysticercus boris it reaches into alimentary canal of man.

The scolex in contact with alimentary canal evaginates and fixes to the gut wall of primary host. This larva fixes by means of a sucker, attaches to the gut wall it absorbs nutrition and develops into a adult worm. The additional proglottids are formed from the neck by the process called strobilization. The worm grows to sexual maturity within 2 to 3 months and start to produce eggs. The gravid proglottid passes out to repeat the life cycle.

## Pathogenicity :-

Taenia saginata causes disease called taeniasis adult worm in the intestine causes various gastro intestinal troubles it includes pain in abdomen chromic indigestion, anaemia, vomiting constipation etc.

The patient also suffers from servere headache & fever eosinophilia may also occur very rarely cysticercus boris get settled in the muscles & various body organs of man. It causes further complication in muscles and organs.

## Diagnosis :-

1) A Taenia infection is easy to diagnosis if the 1.5 – 2 cm long & 0.7 cm wide segments are eliminated in stool.
2) Morphological species differentiation is often not possible based on the gravid proglottids, but can be done by DNA analysis (PCR).

3) T. saginata eggs are shed irregularly in stool and can not be differentiated morphologically from T. solium eggs.
4) Using an ELISA, coproantigens are detectable in stool fluid even when neither proglottids nor eggs are being excreted.

## Prophylaxis :

1) Personal hygiene.
2) Sanitory measures.
3) Strict inspection of pork in slaughter house.
4) Thorough cooking.
5) Proper disposal of human faeces.
6) Avoid eating raw vegetables grown on woil irrigated by sewage water.
7) Treatment of infected person.

## Treatment :-

1) Treatment of patient with antiheminthes drugs to remove adult worm.
2) The patient is treated with ccl4 tetrachloro ethylene, for cerebral cystacereosis. Such drugs are available in every where the drugs.

## 2) TAENIA SOLIUM :-

### Systematic position :-

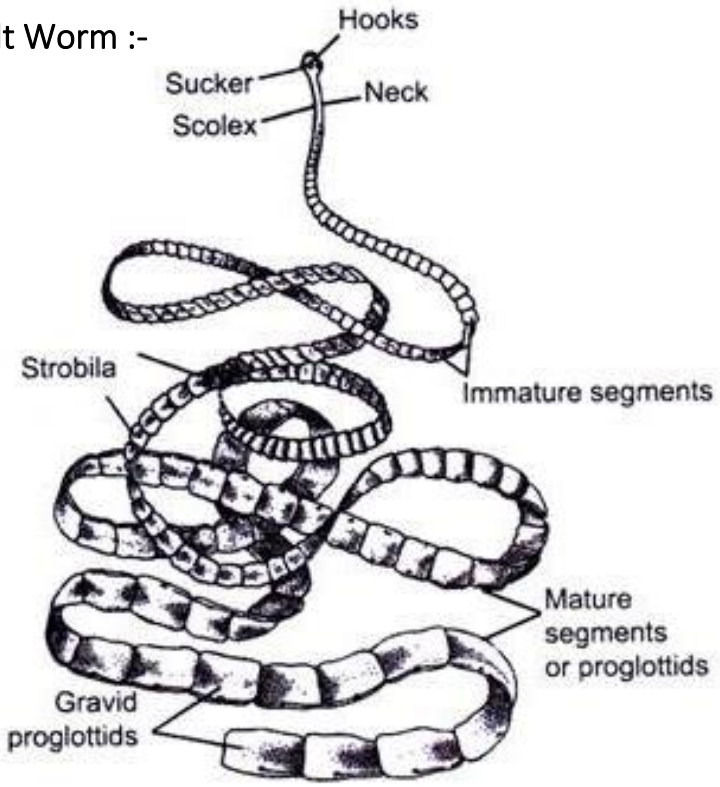
Phylum - Platyhelminthes
Class - Cestoda
Order - Clyclophllidea
Family - Taeniidae
Genus - Taenia
Species - T. Solium

### Geographical Distribution :-

Common names for Taenia Solium are “The pork tapeworm” and “The armed tapeworm”. It is also world wide in distribution. The infection occurs by eating raw or insufficiently cooked pork. It is uncommon in Mohammedans who are not generally pork-eaters.

- **Morphology :-**

### 1) Adult Worm :-



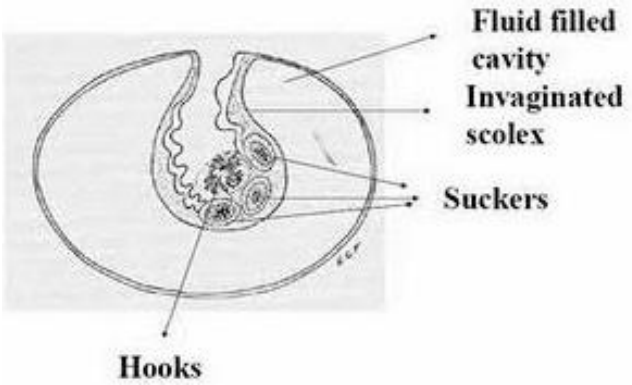
The adult Taenia solium has a head (scolex) that consists of four suckers and a rostellum with a double crown of hooks and unsegmented narrow neck and a large body formed by several hundred proglottids.

The entire body is called a strobila and may range in size from two to eight meters long. Each proglottid of Taenia solium has reproductive organs from both sexes and a complex absorption & reproduction system. Proglottids that are closer to the scolex do not have sexual organs. The further from the scolex the more sexually mature the proglottid mature proglottid contains more than 50,000 fertile eggs which are accumulated with a long and profusely branched central uterus.

Taenia solium dwells within the small intestine of humans where it is attached to the intestinal wall with the powerful suckers & the hook. Every few days 2-5 gravid proglottids, each measuring about 5x2 cm are detatched from the distal end of the body of the worm & are passed with feces.

Each proglottid liberated thousands of eggs which are fully embrionated, infective and very resistant to adverse environments; eggs can remain viable for up to two months in water soil & vegetation particularly in humid and warm environments.

2) Cysticercus :-



Taenia solium cysticercus is a liquid filled vesicle measuring from 10 to 20 mm. in diameter with great variation in size. It consist of the vesicular wall and the invaginated scolex a profuse network of excretory and neural stru. is also seen within the vesicular wall. Taenias and cysticerci fulfil their metabolic & nutritional needs by absorption & diffusion through the vesicular wall. The vesicular fluid monstly composed of water although it also contains calcium glycoproteins etc.

It may be carried in the blood stream to any part of the body & may lodge in any tissue, but it most frequently develops in the voluntary muscle.

The larva completes developments in about 2 months. It is semitransparent opalescent white & elongate oval in shape & may reach a length 0.6 to 1.8 cm. The bladder is fluid filled & on one side is a denser area containing the scolex.

## Life-Cycle :

The life cycle of T. Solium is indirect. It passes through pigs or other animals, as intermediate hosts, into humans as definitive hosts. In humans the infections can be relatively short or long lasting and in the latter cases if reaching the brain can last for life from humans, the eggs are released in the environment where they await ingestion by another host. In the secondary host, the eggs develop into oncospheres which bore through the intestinal wall & migrate to other parts of the body where the cysticerci form. The cysticerci can survive for several years in the animal.

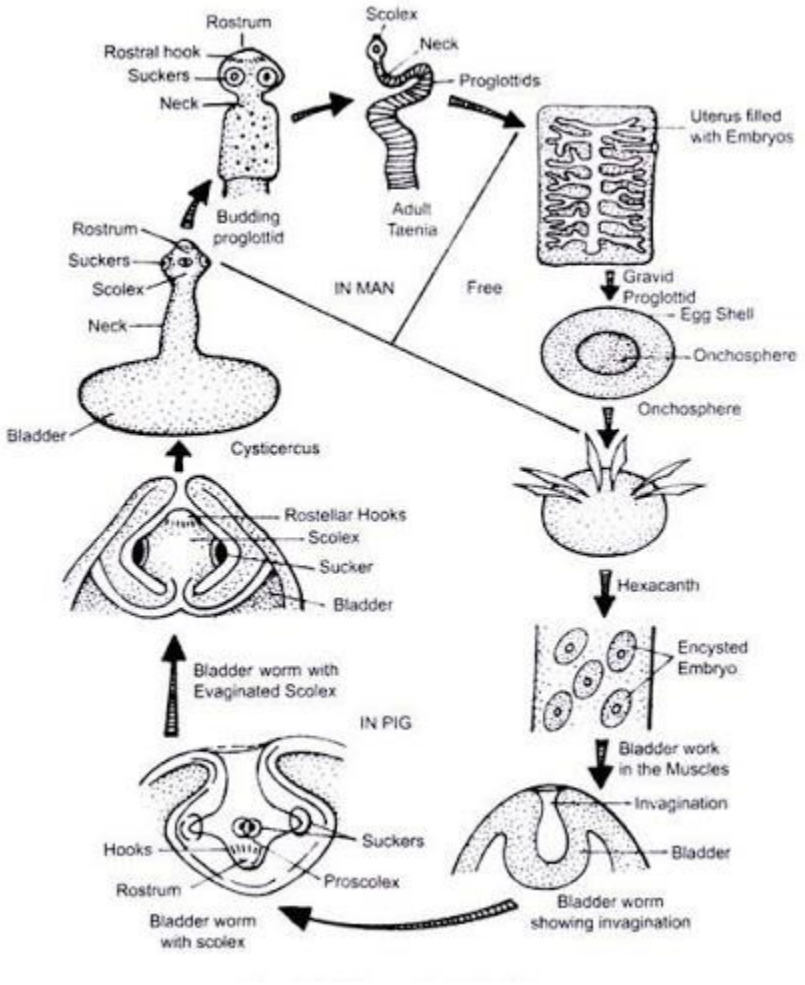



***Fig. Life Cycle T. Solium***

## Definitive Host :-

Humans are colonized by the larval stage the cysticercus, from undercooked measly pork or other meat. Each microscopic cysticercus is oval in shape, containing an inverted scolex, which everts once the organism is inside the small intestine. This process of evagination is stimulated by bile juice & digestive enzymes (of the host).

Using the scolex, it anchors to the intestinal wall. It grows in size using nutrients from the surroundings. Its strobila lengthens as new proglottids are formed at the foot of the neck. In 10-12 weeks after initial colonization. It is an adult worm.

As a hermaphrodite, it reproduces by self-fertilization, or cross-fertilization if gametes are exchanged between two different proglottids. Spermatozoa

fuse with the ova in the fertilization duct, where the zygotes are produced. The zygote undergoes holobastic & unequal cleavage resulting in three cell type small medium & large (micromeres, mesomeres, megameres).

Megameres develop into a syncytial layer, the outer embryonic membrance; mesomers into the radially striated inner embryonic membrane or embryophore; micromeres become the morula. The morula transforms into a six-hooked embryo known as an oncosphere or hexacanth larva.

A gravid proglottid can contain more than 50,000 embryonated eggs. Intact gravid proglottids are shed off in groups of four or five ionospheres can survive in the environment for up to two months.

## Intermediate Host :-

Pigs are the most common host who ingest such eggs in traces of human faces, mainly from vegetation contaminated with it such as from water bearing traces of it. The embryonated eggs enter intestive where they hatch into motile oncospheres. The embryonic and basement membranes are removed by the hosts digestive enzymes (particularly pepsin). Then the free oncospheres attach on the intestinal wall using their hooks. With the help of digestive enzymes from the penetration glands, they penetrate the intestinal mycosa to enter blood & lymphatic vessels.

They move along general circulatory system to various organs & large numbers are cleared in the liver. The surviving oncospheres preferentially migrate to striated muscles as well as the brain, liver & other tissues where they settle to form cysts – cysticerci. A single cysticeccus is spherical, measuring 1-2 cm in diameter & contains are invaginated protoscolex. The central space is filled with fluid like a bladder hence it is also called bladder worm cysticerci are usually formed within 70 days & may continue to grow for a year.

Humans are also accidental secondary hosts when they are colonized by embryonated eggs, either by auto-colonization or ingestion of contaminated food. As in pigs, the oncosphere hatch & enter blood circulation. When they settle to form cysts, clinical symptoms of cysticercosis appear. The cysticercus is oftem is called metacestode.

Pathogenicity :-

They usually do not give rise to any symptom. Occasionally vague abdominal discomfort chronic indigestion, anemia and intestinal disorders may be present.

Cysticercus cellulose may develop in any organ. They usually develop as visible nodules in the subcutaneous tissue & muscles. It may also develop in brain leading to epileptic attacks.

- Diagnosis :-

i) Intestinal Taeniasis :-

a) Stool Examination :- Naked eye examination of the stool should be made for segments of Taenia SP. A microscopic examination of the specimen for the eggs can be carried out by direct smear & concentration method sedimentation technique.

b) Molecular Methods :- DNA probes and PCR have been developed to detect and differentiate between eggs of T. solium in stool specimens.

ii) Diagnosis of Cysticercosis :-

1) Biopsy examination of subcutaneous nodules for cysticerci.
2) X-ray of skull & soft tissues to reveal calcified cysticerci.
3) CT scan is the best method for deteching calcified cysts.
4) MRI is superior to CT scan for demonstration of non-calcified cysts.
5) Eosinophilid.
6) Antigen can be detected in the serum or CSF by ELISA.
7) A history of intestinal taeniasis is another aid in the diagnosis.

- Prophylaxis :-

1) Avoidance of eating raw or under cooked meat.
2) Avoid eating raw vegetables grown on soil irrigated by sewage water.

3) Effective treatment of infected individual to prevent infection of the intermediate hosts.

4) Personal hygiene to prevent the risk of acquiring cysticercosis by autoinfection.

- Treatment :-

1) Praziquantel and niclosamide are used for the treatment of human tapeworm infection.

2) A single dose of 2.0 gm of (four tablets of 500 mg) niclosamide is effective against adult worms of T. solium in the intestine. However, praziquantel is the drug of choice for the treatment of T. solium infection because it not only kills the adult tapeworm in a single dose, but also kills the cysticer when taken in high dose for 3 to 7 days.

## 3) ECHINOCOCCUS GRANULOSUS :-

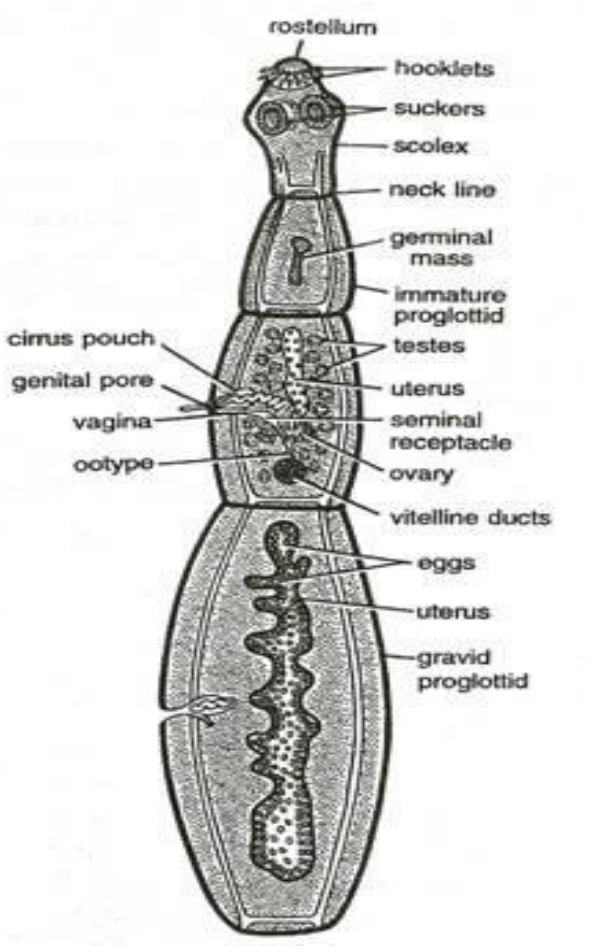
### Systematic position :-

Phylum - Platyhelminthes
Class - Cestoda
Order - Clyclophllidea
Family - Taeniidae
Genus - Echinococus
Species - E. granulosus

### Geographical Distribution :-

Although the hytid disease is world wide in distribution. It is most commonly found in those countries where sheep and cattle raising areas and consequently there is a close association between man, sheep & dog. It has been reported from South Australia, Argentina, China, Mangolia, Japan, Arabia, India (in Punjab) & USA.

- **Morphology :-**



It is commonly called as 'Dog tape worm' it is common in man and domestic animals it belongs to –

It is very small in size. It measures up to 2-8 mm in length. Its body divisible into three parts such as –

(1) Scolex or head.

(2) Neck.

(3) Large proglottids.

### 1)Scolex or head :-

The anterior end of the body consist of a knob like scolex, which is 500 mm. in diameter. It is muscular. It is provided with suckers. It shows a rostellum with two rows of hooks.

### 2)Neck :-

The narrow part of the scolex behind the suckers from the neck it is a proliferating zone. It is thick.

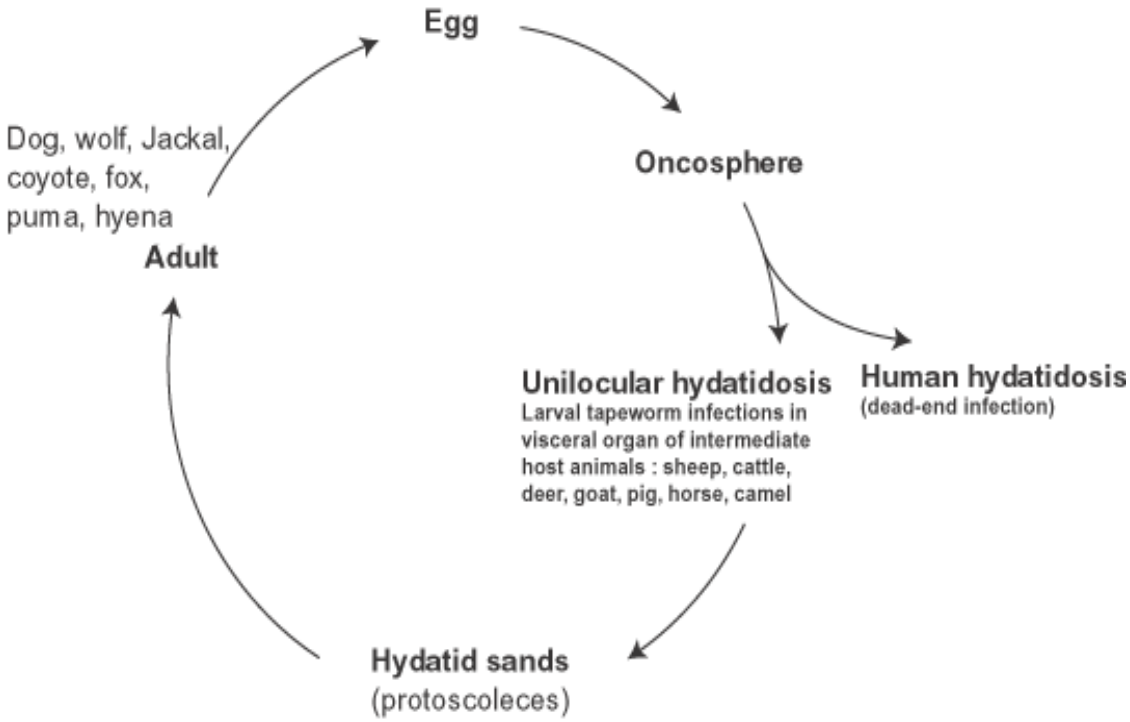
### 3)Strobilla :-

It shows three proglottids, first one is immature. The second is mature proglottid. The third one is gravid proglottid.

The second and mature proglottid will show completely developed reproductive organs. It is hermaphrodite. The male reproductive system shows 20-30 testes. The vas differences is coiled tube. It opens into genital chamber. The female reproductive system includes ovary, oviduct, vagina, uterus, vitellaria Ootype (surrounded by shell glands).

The third proglottid is gravid proglottid is filled with fertilized eggs.

- Life-Cycle :-




It is a bisexual animal. It is a digenetic parasite. Its life cycle completes within two hosts.

Definite host is dog while the secondary or intermediate host is man and domestic animals like a goat, dog, rabbit etc.

## Modes of Transmission :-

The gravid proglottid contains branched uterus. It is filled with eggs. They are nearly 500-800 in numbers. The gravid proglottid ruptures and the eggs are liberated into the intestine. Each egg is 30 micron in length & 15 micron in width. These eggs will come out with fecal matters of dog.

## Transmission in the Secondary Host (Man) :-

Here secondary host is man, herbaceous mammals like sheep rabbit, cattles etc. when the secondary host taken contaminated water or food the anchosphere will enters into the gut of the secondary host.

In the duodenum of the secondary host the shell is dissolved. The embryo is liberated. It contains, six hosts with the hooks it penetrates into the blood vessels, through the intestinal wall. Then it reaches wings, liver, brain, heart or eye and develops into hydatid cyst.

## Hydatid Cyst :-

When the embryo reaches lung, liver, heart, brain it becomes large and becomes a waterly bladder. Around the bladder the tissue of host will develop a cyst. It becomes a hydatid cyst. The cyst contains outer pericysts middle ectrocyst & inner endocyst.

In man hydatid cyst grows slowly they are round it may grow to the size of foot ball. The different kinds of hydatid cyst are recognized as –

(a) Unilocular hydatid cyst.
(b) Multilocular hydatid cyst.
(c) Alevolar hydatid cyst.

## Modes of infection to man :-

When man is closely associated with the dogs the eggs may find always into the body of man. In the human beings kiss the dog or allow the dog to kiss them or allow the ug to contaminate their food and water then man may infected with eggs.

## Pathogenicity :-

When the cyst grows into big size the surrounding tissue will develop fibrous tissue and the organs system enlarge – for ex –

1) If the cysts are present in the river, it becomes very big.
2) If the cyst are present in the brain it may be featal to man.
3) If the fluid of the cyst enters into the body of the host it causes pain diarrhea.

## Diagnosis :-

i) **Casoni test :-** It is an immediate hypersensitivity skin test introduced by casoni in 1911 sterile hydatid fluid is used as antigen. The hydatid fluid is drawn from unilocular hydatid cysts from human cases or from animals. It is filtered & made sterile.

ii) **Blood Examination :-** Eosinophilia (20-25%) may be present.

iii) **Serological tests :-** Enzyme-linked immunosorbent assay complement fixation test indirect haemagglutination assay & latex agglutination tests are used for diagnosis of hydatid cysts.

iv) Examination of cyst fluid.

v) Radiological examination.

vi) Histological examination.

**Prophylaxis :-**

E. granulosus infection can be prevented by –

1) Not allowing the dogs to eat carcases of slaughtered animals in endemic areas.

2) Improvement in personal hygiene.

**Treatment :-**

Surgical removal of the hydatid cyst can be performed but there may be recurrences in 2 to 25% cases after surgery.

Hence, postoperative chemotherapy may be given for at least two years after surgery. Praziquantel & albendazole are the chemotherapeutic agents used for the treatment of hydatid cyst.

## 4) HYMENOLEPIS NANA :-

### Systematic position :-

Phylum - Platyhelminthes

Class - Cestoda

Order - Clyclophllidea

Family - Hymenolepididae

Genus - Hymenolepis

Species - H. nana

### Geographical Distribution :-

The common name used for H. nana is the dwarf tapeworm. It is the smallest tapeworm infecting humans. The name Hymenolepis (hymen meaning membrane, lepis meaning covering) means a thin membrane covering the egg and nana (nanus – dwarf or small) means small size. It is more common in warm than in cold climates.

- **Morphology :-**

### 1) Adult Worm :-

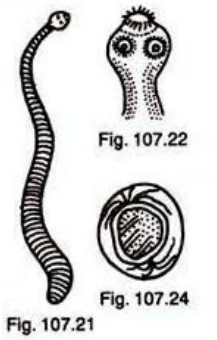


Fig. 107.21



Fig. 107.22



Fig. 107.24

Fig. 107.25

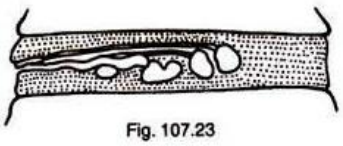


Fig. 107.23

The adult worm is small measuring only 4 to 5 cm in length & 1 mm in diameter. Like other tapeworms, it consists of scolex, neck and strobila.

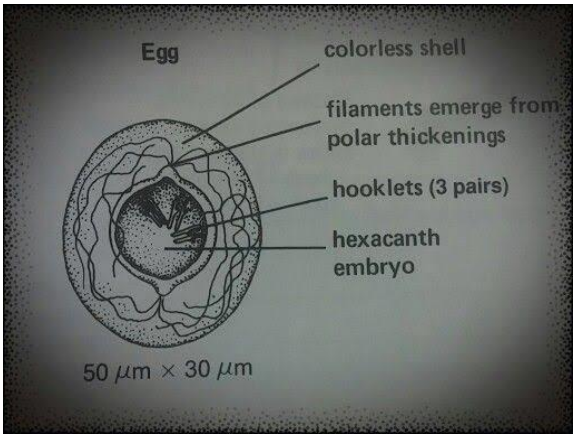
**Scolex :-** It is globular in shape and has four suckers. It contains a retractile restalum with a single row of hooklets.

**Neck :-** It is long, slender and is situated posterior to the scolex.

**Strobila :-** The neck is followed by the strobila consisting of 200 or more proglottids. These proglottids are much broader than long.

Life span of the adult worm is about two weeks.

**Egg :-**




It is spherical or ovoid, 30 to 45 μm in diameter. If contains a smooth thin colourless outer membrane and an inner membrane (empryophore) enclosing a hexsacanth embryo (oncosphere).

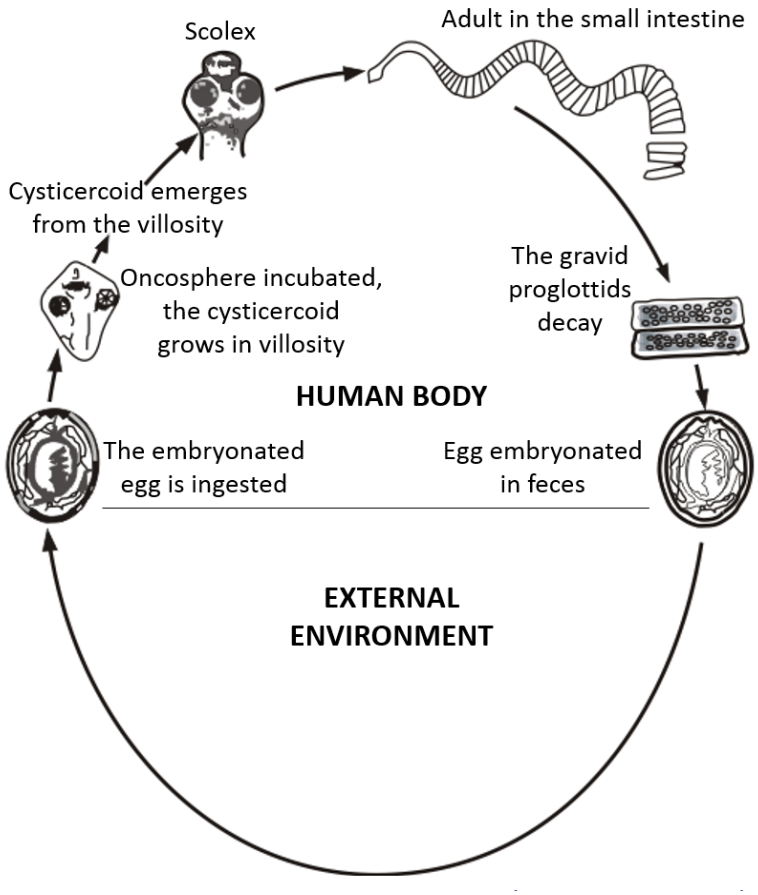
The space between the two membranes contains yolk granules & 4 to 8 polar filaments arising from two knobs on the embryophore. The eggs floats in saturated salt solution & is non-bile stained.

**Life-Cycle :-**

H. nana is the only cestode which completes its life cycle in single host. Adult worm resides in the ilcum of human beings.

Infection occurs by ingestion of the eggs by faecal-oral transmission. In the small intestine a free oncosphere (hexacanth embryo) is liberated from the egg. The hexacanth embryo penetrates into the villus of small intestine & develops into the cysticercoids larva in about 5 days. Later on the villus

repures and the cysticercoids larva becomes free in the lumen of small intestine. It attaches by its scolex to another villus further down. In course of two weeks or more it develops into an adult worm.




It start strobilisation and in about 30 days after the infection, the eggs and proglottids begin to appear in faeces and the life cycle is repeated. The egg may hatch out in the intestine before passing out in faeces liberate embryos which invade intestinal wall. This result in internal autoinfection. It is responsible for increase in the worm load of the intestine. Another type of autoinfection is external autoinfection in which a person ingests one's own eggs by faeco-oral route due to bad personal hygiene. This result in persistence of infection in a host.

A different strain of H. nana may infect rats & mice. The eggs of H. nana are passed in the faeces of these rodents. Rat fleas ingest the eggs of H. nana. These insects act as intermediate host. The egg develop into cysticercoids larva in the body cavity of these insects. Rodents get infected when they eat these insects. The nurine strain of H. nana does not appear to infect humans. However, the human strain may infect rodents, which may this constitute a reservoir of infection for human parasite.

Pathogenicity :-

The infection with H. nana does not generally produce any disease. Symptoms produced are due to allergic reaction.

These include abdominal pain, diarrhea & prutitis. The infection is more common in children.

Doagempsos :-

The diagnosis is made by demonstration of characteristic egg in faeces by direct microscopic examination. Concentration methods such as salf floatation & formal-ether may be used for demonstration of these eggs.

Prophylasix :- Preventive measures in include -

1) Personal hygiene.
2) Sanitary improvements.
3) Avoid contaminated food & water.
4) Rodents control.

Treatment :-

Praziquantel is the drug of choice. A single dose is highly effective. Miclosamide may also be used as second choice.

## III) Parasitic Adaptations in Cestodes :-

Parasites are those living with expances of the another living organism. Thus the parasites may be defined as the those species which exists expances of certain other species called host-parasites are biologically & economically closely connected with host throughout their life. This association is called parasitism parasite live in the body of host. They have to modify their body structurally & physiologically. This change & modification in the body of parasite for parasitic mode of the life is called parasitic adaptation.

Adaptation is fitness of an organism to it's environment the parasitic nematodes. Cestodes and trematodes particularly have undergone profound adaptation the suit their parasite mode of life as it is stated that their adaptation are morphologically as well as physiologically forms.

### A) Morphological Adaptations :-

#### i) Body Covering :-

The body of parasite is covered by a thick covering called "tegument". A tegument is also with scale to give protection to the parasite. The tegument is probably a protoplasmic lager but continuously and renewed by cells.

#### ii) The Shape of body :-

Generally the shape of body is small flat dorso ventrally flattened leaf like or ribbon like as it is found in most of cestodes.

#### iii) Organs of body :-

For the grip and the attachment to the body of host & tissue parasites have some special organs of attachment.

The flatworm like cestodes for this purpose are various arms with suckers, hooks & spines. Then sucker may be with or without hoosk & spines.

### iv) Organs of Locomotion :-

Parasites are without organs of locomotion because parasites leave in the body of host where sufficient food is available without any effort. Thus the organs of locomotion such as cilia, flagella etc. are absent. Without any effort and movement they can even reproduce, because other opposite sex individuals are available.

### v) Organs of nutrition :-

Being parasite mode of nutrition the food of the parasite is already digested food semi digested forms.

In cestode parasites the food is directly absorbed by the general surface of the body.

### vi) Neuro Sensory System :-

It is required by the free living animals for quick & efficient response to stimulate. But it is not required for the parasites parasitic life is in a scale environment. Hence there is a profound reduction of nervous system & the total absence of sense organs.

### vii) Reproductive System :-

In cestodes on the other hand reproductive system is much more elaborate, each mature proglottid possese complete set of male & female genitalia. In gravid proglottids all other organs of system degenerate to make space for the uterus which becomes highly enlarged & branched to accommodate a large number of eggs. In addition to the enormous development of reproductive organs.

There are various glands associated their secretion helps in the formation of protective covering over the fertilized eggs and also helps in unbricatting the various parts of the system to pass eggs easily.

### B) Physiological Adaptations :-

### i) Protective mechanism :-

The alimentary canal of parasite and the body are variously modified to protect themselves from the action of digestive juice. In tapeworm gut wall secretes which forms protective covering around the parasites, trematodes & cestodes also secretes antienzyme to neutralize the digestive enzyme of the host. The other covering of the parasite is called cuticle or integument. If the tough hand protective.

### ii) Anaerobic respiration :-

Parasite respires anaerobically by breaking down glycogen because environment in the gut & various parts of the body of host is without free oxygen.

### iii) Osmoregulation :-

In the cestodes the osmotic pressure is little higher which permit ready obsorption of digested food by the parasite.

### iv) High fertility :-

Fertilized egg produced by parasites have very uncertain features. It is due to complex life histories hence. The parasite are producing large number of ova to bring about fertilization and larger number of fertilized egg are produce through some of the fertilized eggs are destroyed. The remaining may develop into adult hood. Thus existence of the species is maintained.

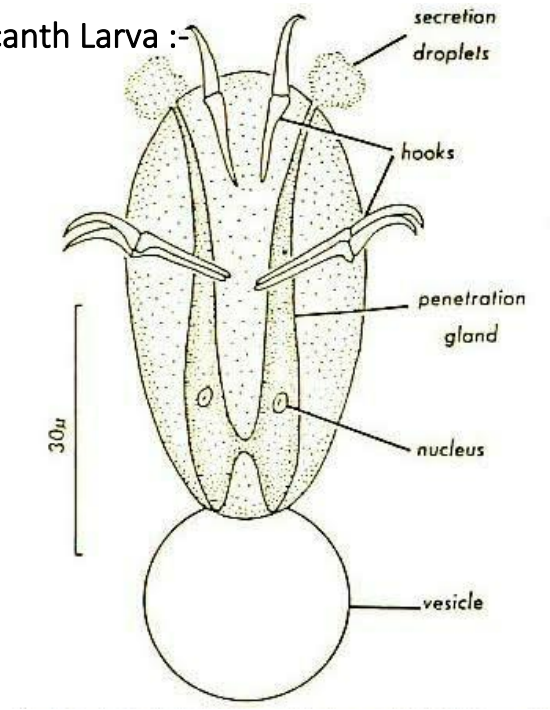
v) Polyembryony or pedogenesis :-

Poly embryony is the process in which asexual multiplication of larval forms occur. In case of various parasitic cestodes the larva are multiplied asexually such as radia gives rise to large was of daughter readia from the large germ balls of the parent radia.

In the same way a single hexacanthe of echinococcus produces several scolices each of which is a potential type worm.

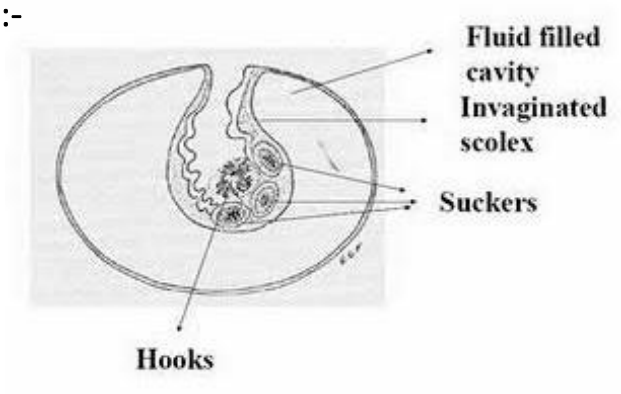
IV) Larval forms in Cestodes :-

1) Hexacanth Larva :-




The motile six-hooked first stage larva of cyclophyllidean cestodes, it emerges from the egg & actively claws its way through the intermediate hosts intestine before development into the next larval stage; for example, the hexacanth of Taenia saginata which penetrates the intestine of a cow that ingested the egg.

Cysticercus Larva :-

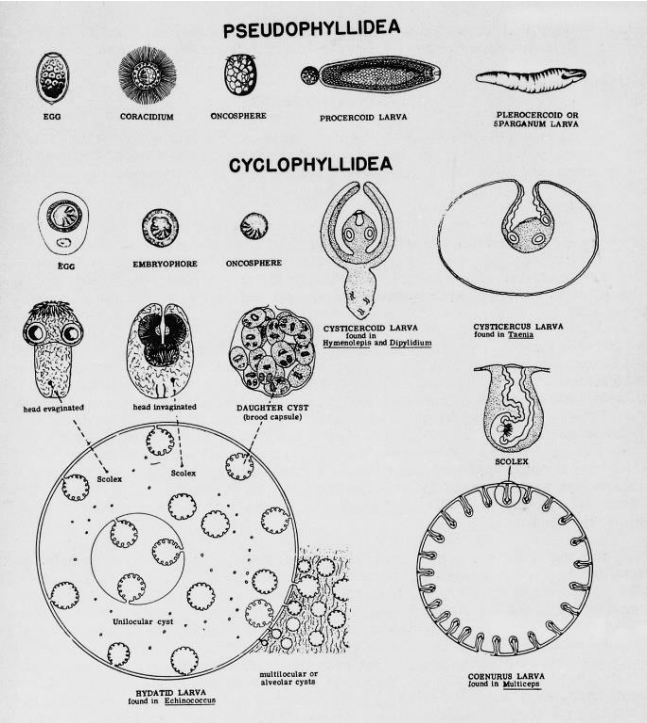



Cysticercus is a scientific name given to the young tapeworms belonging to the genus Taenia. It is a small sac-like vesicle resembling a bladder hence, it is also known as bladder worm. It is filled with fluid, in which the main body of the larva called scolex.

It normally develops from the eggs, which are ingested by the intermediate hosts, such as pigs & cattle the tissue infection is called cysticercosis. Inside such hosts, they settle in the muscles. When human eat reaw or under cooked pork or beef that is contaminated with cysticer the larva grow into adult worms inside the intestine.

Under certain circumstances, specifrically for the pork tapeworm, the eggs can be accidentally eaten by humans through contaminated foodstuffs. In such case the eggs hatch inside the body, generally moving to muscles as well as inside the brain. Such brain infection can lead to a serious medical condition called neurocysticercosis. This deasease is the leading cause of acquired epilepsy.

2) Oncosphere Larve :-



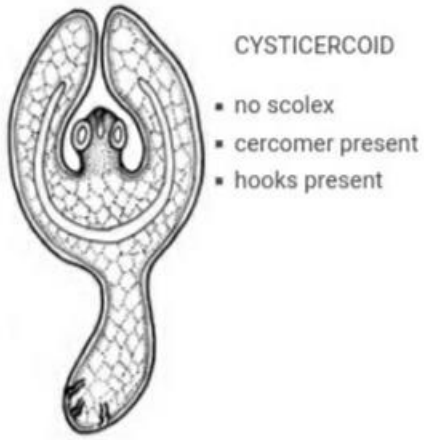

The oncosphere (hexacanth embryo) possesses three pairs of small hooks. In the direct life cysle embryonated eggs are ingested by the definitive host & hatch in the small intestine, where the oncosphere emerges and penetrates into villi to develop into a cysticercoids arva in 4 to 5 days.

The bladderwom is a cyst created by the oncosphere.

An oncosphere is the larval forms of a tapeworm once it has been ingested by an intermediate host animal.

The worm will grow in length & eventually produce proglottids which will exist the intestinal tract with other waste material & then burst releasing the worms eggs & completing the cycle.

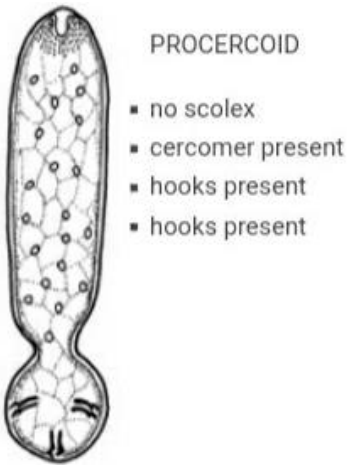
3) Cysticercoid Larva :-




A cysticercoids is the larval stage of certain tapeworms, similar in appearance to a cysticercus, but having the scolex filling completely the enclosing cyst.

In tapeworm infections, cysticercoids can be seen in free form as well as enclosed by cysts in biological tissue such as the intestinal mucosa.

4) Procercoid Larva :-

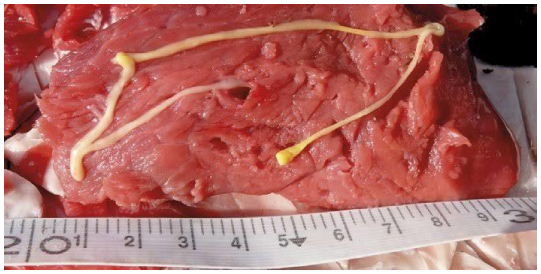


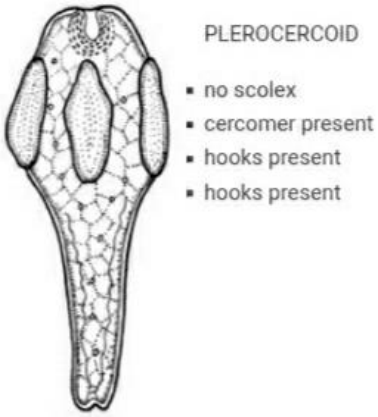

Procercoid is the first larval stage of some tapeworms, which usually develops inside the body cavity of copepods.

The flatworm in this stage is not enclosed in a protective cyst but is infectious.

Procercoids resemble their adult forms in pathway of energy metabolism.

## 5) Plerocercoid Larva :-






Plerocercoid larvae of spirometra SP. Isolated from muscle tissue of wild boar hunted in Bialowiez primeval forest Poland. The average length of sparganum was 103 mm crange 30-290 mm.

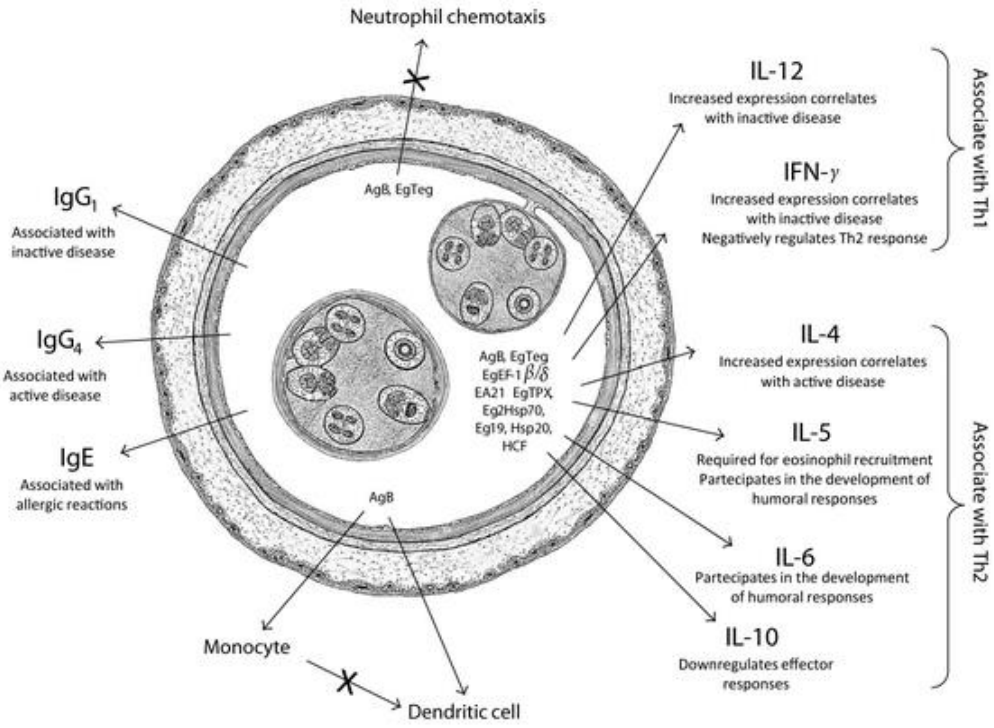
The order to rule out lung cancer, we peformed a VATS bipsy in the right upper lower lobes & pleura a sparaganum larva was seen migreating along the parietal pleura.

The serum ELISA taken seven days after surgery was positive for anti sparganum antibody.

## 6) Sparaganum Larva :-

1) Sparaganosis is a parasitic infection caused by the plerocercoid larva of the genus spirometra including S. mansoni S. ranarum, S. mansonoides and S. erinacei.
2) It was first described by Patrick manson from china in 1882 & the first human case was reported by Charles wardell stiles from florida in 1908.
3) The infection is transmitted by ingestion of contaminated water ingestion of a second intermediate host such as a frog or snake, or contact between a second intermediate host & an open wound or mucous membrane.
4) Humans are the accidental host in the life cycle, while dogs, cats & other mammals are definitive hosts.

7) Hydatid cyst Larva :-



1) The cyst is lined by a multilayer parasite tissue with the innermost layer being the germinal layer.
2) GL can spawn the formation of brood capsules which are themselves lined by GL.
3) the daughter cysts (the encircled body) into the center of the fluid-filled cyst.
4) They will develop into tapeworms should this be eaten by a definitive or final host such as a canine.
5) Found within the hydatid cyst developing inside various organs of the intermediate host.
6) Represents the structure of the scale of adult worm & remains invigilated within a vesicular body.
7) Human are infected by ingesting eggs shed in the feces of canine definitive host.
8) The result is the formation of slow growing larval cyst in the liver, lungs, or other organ systems, which eventually produce clinical signs from mass effects allergic reactions or through tissue necrosis fibrosis.

# DEFINATIONS

Parasite: living organism requiring intimate prolonged contact with another living organism to meet some of its basic nutritional needs. In a more restricted definition, it refers to organisms that are not viruses, bacteria, fungi, rickettsia, or Chlamydia and obviously include organisms of varying complexity from unicellular protozoa to complex multi cellular helminths.

Host: organism harboring a parasite.

Definitive host: animal harboring the adult or sexually mature stage of the parasite.

Intermediate host: is one that is required for parasite development but one in which the parasite does not sexual maturity .

Life cycle: for survival and reproduction reasons many parasites evolve through a number of morphologic stages and several environments or different hosts. The sequence of morphologic and environmental stages is referred to as the life cycle.

Parasitic infection: invasion by endoparasites (protozoa and helminths).

Parasitic disease: invasion and pathology produced by endoparasites.

Parasitic infestation: external parasitism by ectoparasites (arthropods).

Commensalism: the association of two different species or organism in which one is benefited and the other is neither benefited nor harmed. (e.g. non pathogenic intestinal protozoa)

Reservoir host: an animal that harbours a species of parasite that can be transmitted to and infect man.

Vector: an arthropod or other living carrier that transports a pathogenic organism from an infected to a non-infected host. This can be passive transport VII or as an essential host in the life cycle of the pathogenic organism (i.e. a biologic vector).

Carrier: a host that harbours a parasite but exhibits no clinical signs or symptoms.

Zoonosis: a disease involving a parasite for which the normal host is an animal, and wherein man can also be infected.

Protozoa: a subkingdom consisting of unicellular eukaryotic(Greek-eu= true,karyon=nut=nucleus) animals.

Eukaryote: a cell with a well-defined chromosome in a membrane bound nucleus (versus prokaryotic bacteria with nucleic acid material bound in a nuclear membrane).

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